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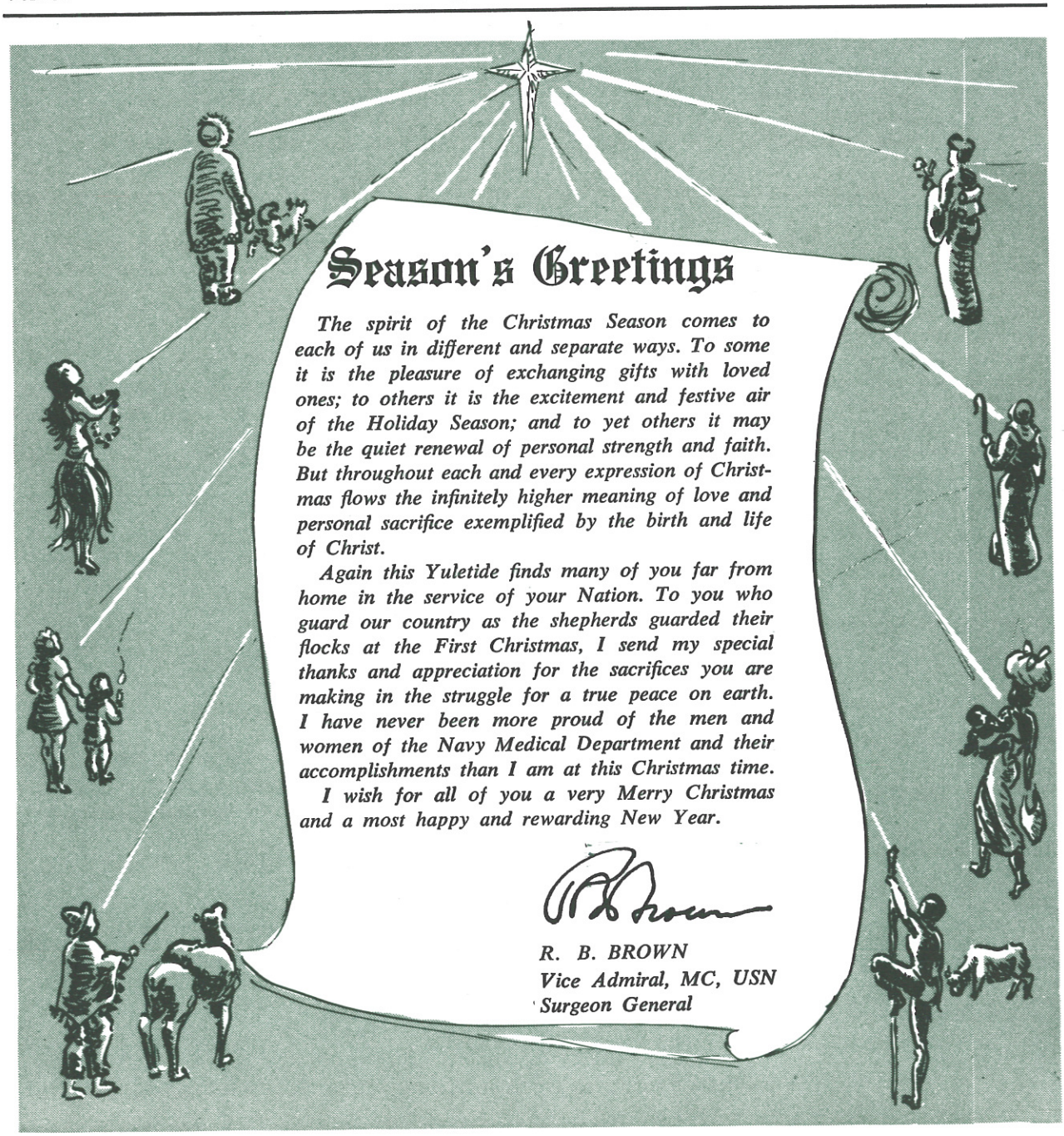

Season's Greetings

The spirit of the Christmas Season comes to each of us in different and separate ways. To some it is the pleasure of exchanging gifts with loved ones; to others it is the excitement and festive air of the Holiday Season; and to yet others it may be the quiet renewal of personal strength and faith. But throughout each and every expression of Christmas flows the infinitely higher meaning of love and personal sacrifice exemplified by the birth and life of Christ.

Again this Yuletide finds many of you far from home in the service of your Nation. To you who guard our country as the shepherds guarded their flocks at the First Christmas, I send my special thanks and appreciation for the sacrifices you are making in the struggle for a true peace on earth. I have never been more proud of the men and women of the Navy Medical Department and their accomplishments than I am at this Christmas time.

I wish for all of you a very Merry Christmas and a most happy and rewarding New Year.

R. B. BROWN
Vice Admiral, MC, USN
Surgeon General

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Policy

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The issuance of this publication approved by the Secretary of the Navy on 4 May 1964.

BACTERIAL ENDOCARDITIS — CHANGES IN THE CLINICAL SPECTRUM

Ralph Tompsett MD, Dallas, *Reprinted from the Archives of Internal Medicine April 1967, Vol. 119, pp. 329–332,*

For many years bacterial endocarditis has seemed to evoke widespread interest among clinicians. Of all the protracted febrile illnesses it has been one of the most challenging as an exercise in diagnostic acumen. The reasons for this have been several. First, the site of infection is impalpable and cannot be visualized even with the most modern diagnostic techniques. Second, the cardinal signs—fever, heart murmur, and emboli—may often be caused by other diseases, or they may be absent in endocarditis. Finally, the nature of embolization is such that lesions may be produced in any organ and readily simulate other disorders. In spite of such widespread clinical interest, however, the disease is sufficiently uncommon and sufficiently complex that it may readily be missed for long periods. For example, in three recently reported series[1-3] the average duration of symptoms prior to initiation of definitive therapy ranged from 2.2 to 5 months.

The purpose of this paper is to review a few of the clinical findings in bacterial endocarditis in order to emphasize some of its well-known features as well as to review some changing concepts about this intriguing febrile disease.

Composition of the Series

The patients to be described were observed by the author during the nine-year period 1957 to 1966. There were 76 episodes of endocarditis in 74 patients. Two patients had reinfections, and one had a double infection. The causative microorganisms were streptococci in 48, staphylococci in 22, and a miscellaneous group in seven. Table 1 gives a listing of the various causative microorganisms. Thirty cases were due to viridans streptococci; five, to various hemolytic streptococci exclusive of group D; and six, due to enterococci. Seven cases were due to other streptococci. Twenty-two were caused by staphylococci; two, by pneumococci; and one each by *Escherichia coli, Histoplasma,* and *Candida.* Two had autopsy evidence of bacterial endocarditis, but cultures were negative. This series, which compares closely with other recent series[3,4] illustrates the fact that nearly half of the cases were due to so-called pyogenic microorganisms. This is an important distinction, for in the diagnosis of obscure fevers, the demonstration of persistent bacteremia due to viridans streptococci is really diagnostic of endocarditis. When pyogenic microorganisms are found, however, one must rely on other diagnostic features because of the possible existence of other sources of the bacteremia such as localized abscess or urinary tract infections.

TABLE 1. *Bacterial Endocarditis—Composition of Series*

Microorganisms	No. of Patients
Streptococci	
Viridans streptococci	30
Hemolytic streptococci	5
Group A-2	
Group B-2	
Non A or D-1	
Enterococci (group D)	6
Other streptococci	7
Total streptococcal	48
Staphylococci	
Coagulase positive	20
Coagulase negative	2
Miscellaneous	
Pneumococcus	2
E coli	1
Histoplasma	1
Canadida	1
Uncertain (autopsy, no positive culture)	2
Total nonstreptococcal	29

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Presented as a part of the Symposium on Mysterious Fevers, Recent Diagnostic and Therapeutic Advances, at the 115th annual convention of the American Medical Association, Chicago, June 28, 1966.

Fever

The most consistent clinical finding was fever. All of these cases have been reviewed for the presence of fever and arbitrarily those cases noted in which the patients had no clear-cut history of fever and no temperature elevations over 100.5F (38.1C) for the first 48 hours in the hospital. This occurred only five times. Two of the five patients were receiving antimicrobial drugs up to the time of admission, and this may account for the absence of fever. One patient had hypothermia associated with a subarachnoid hemorrhage. In two, no obvious explanation was at hand. Curiously enough both of these had staphylococcal endocarditis. It must be added that in all but one of these five, fever was present on at least one day during the first week, and thus fever was a most reliable sign.

Murmurs

It was of particular interest to review data on the presence of heart murmurs. These patients were listed on the basis of the first thorough and carefully recorded cardiac examination either subsequent to knowledge of the presence of bacteremia or when it was clear that endocarditis was being considered by the examiner. A summary of the data is presented in Table 2. In 16 of the 76 cases no murmurs were recorded. Among 30 patients with viridans streptococcal disease, only two had no murmurs. No murmur was heard in two of the seven patients with nonhemolytic streptococci, in three of six with enterococci, and in five of 20 with infections due to coagulase positive staphylococci. These data are in accord with previous observations that these so-called pyogenic microorganisms are capable of attacking normal heart valves and hence absence of murmurs might be expected more commonly in this group of patients. The frequency of this finding, however, was surprisingly high. The one patient with *Histoplasma* infection never had a murmur. This is often the case in fungal infections, an observation presumably related to the presence of vegetations which are large in comparison with those caused by bacteria. Table 3 gives follow-up observations in these patients. Ten of the 16 developed murmurs, nine within the first two weeks. As indicated in Table 3, only three patients have been observed over long periods without developing murmurs. Thus, although murmurs are fairly frequently absent in the early phases of the disease, they nearly always develop in patients who survive.

TABLE 2. *Patients With Endocarditis Who Had No Heart Murmurs on Admission to Hospital*

Infecting Microorganism	No. of Patients Without Murmur	Total Patients
Viridans streptococci	2	30
Nonhemolytic streptococci	2	7
Enterococci	3	6
Staphylococcus (coagulase positive)	5	20
Miscellaneous	4	7
Histoplasma (1)		
E coli (1)		
Unknown (2)		

TABLE 3. *Follow-Up Observations on 16 Endocarditis Patients Without Murmurs on Admission*

Patients who developed murmurs	
In first week	4
In second week	5
In fifth week	1
Patients who did not develop murmurs	
Duration of Observation	
Died	
After 1 day	
After 2 days	
After 8 days	
Alive	
3 mo later	
2 yr later	
3 yr later	

Unusual Mode of Presentation

Of special interest from the standpoint of diagnostic intrigue are those patients who present themselves with seemingly well-defined clinical syndromes which direct attention away from the heart. In contrast with the usual fevers of unknown origin, these patients appeared initially to have a well-defined cause for fever. These various disease patterns in this series are listed in Table 4. Heading the list are the cases of meningitis, with or without evidence suggesting brain abscess. Three of these were in patients with staphylococcal infection, and one was a patient with autopsy evidence of endocarditis but no positive cultures. Pelvic infection dominated the clinical picture in three. Strokes and "pneumonia" each occurred in two. Suppurative arthritis and phlebitis occurred in one each. Others were a possible example of osteomyelitis and one patient whose first symptoms were those related to subarachnoid hemorrhage. The initial symptom in the patient with *Histoplasma* endocarditis was pain

TABLE 4. *Bacterial Endocarditis—Unusual Modes of Presentation*

Presenting Syndrome	No. of Patients
Brain abscess or meningitis, or both	4
Postpartum or postabortal pelvic infection	3
Stroke	2
Pneumonia	2
Suppurative arthritis	1
Suppurative phlebitis	1
Possible osteomyelitis	1
Subarachnoid hemorrhage	1
Femoral artery occlusion	1

due to a sudden femoral artery occlusion. These are representative of the confusing features sometimes encountered, and the list could certainly be greatly expanded by the individual cases in various published series.

Persistence of Bacteremia

As previously indicated, the pitfalls in the diagnosis of bacterial endocarditis are sufficiently numerous that one relies heavily on blood cultures to confirm the diagnosis. The frequency with which the pyogenic microorganisms are now seen always necessitates the consideration that the source of bacteremia may be a site of infection other than the heart. It has been a clinical impression that when bacteremia in endocarditis is demonstrable, it is persistent, in contrast with the intermittent bacteremia of infection elsewhere. Evidence related to this was obtained by examining the culture data in patients admitted to the hospital in a period of eight years. The survey included all patients who had a clinical diagnosis of endocarditis and in whom at least one blood culture was positive. A total of 57 patients was reviewed and the data are summarized in Table 5. Forty-eight had received no antibiotics within 48 hours of the first culture. In this group, 168 cultures were taken and only six were negative. Nine patients had received antibiotics within 48 hours. In this group, 28 cultures were taken and six were negative. Thus, these figures support the concept that bacteremia, when present in endocarditis, is persistent. Moreover it was surprising to see how little bacteremia was influenced by the admittedly inadequate chemotherapy given before admission. No consideration is given here to those suspected cases in whom no positive cultures were obtained.

Comment

At the present time antimicrobial agents are available with the potential for achieving bacteriologic cure in upwards of 95 percent of patients with bacterial endocarditis. Despite this fact there remains a very significant amount of death and disability due to this disease. In part, this may be explained by features of the disease which we lack the knowledge to control. In part, however, delayed recognition of the disease is still an important factor, and this points up the need for continued awareness of the varied clinical features which characterize bacterial endocarditis.

TABLE 5. *Blood Cultures in Bacterial Endocarditis* *

No. of Patients	Antibiotic Therapy Within 48 Hours	No. of Cultures	No. Negative
48	No	168	6
9	Yes	28	6

* Results in 57 patients each of whom had at least one positive culture.

The present report and other recent reviews suggest the occurrence either of a change in the clinical spectrum of bacterial endocarditis or a change in its recognition.[5] Of special interest are (1) the frequency in the older age groups, (2) the variety of modes of presentation, (3) the number of patients who have no cardiac murmurs at the onset of the disease, and (4) the frequency of microorganisms other than viridans streptococci.

The apparent increase in numbers of cases due to pyogenic microorganisms poses both a diagnostic and a therapeutic problem, accounting for many of the missed diagnoses and for an impressive proportion of the mortality.

Summary

Bacterial endocarditis continues to present intriguing diagnostic challenges. This paper reviews some of the clinical features of a current series of 76 episodes of endocarditis in 74 patients. The pyogenic microorganisms now make up a high proportion of cases. Fever is the most consistent finding. Murmurs are absent in a significant number of patients when they are first examined but generally develop during continued observation. The majority of patients without murmurs in the beginning are infected with microorganisms other than viridans streptococci. The interesting variety of clinical syndromes precipitating hospital admission is discussed. Data on blood cultures are presented in support of the thesis that in endocarditis when bacteremia is demonstrable at all, it is persistent.

References

1. Geraci, J.E.: The Antibiotic Therapy of Bacterial Endocarditis, *Med Clin N Amer* 42:1001, 1958.
2. Tompsett, R.: Bacterial Endocarditis, *Texas J Med* 57:261, 1961.
3. Blount, J.G.: Bacterial Endocarditis, *Amer J Med* 38:909, 1965.
4. Lerner, P.I., and Weinstein, L.: Infective Endocarditis in the Antibiotic Era, *New Eng J Med* 274:199–206, 1966.
5. Uwaydah, M.M., and Weinberg, A.N.: Bacterial Endocarditis—A Changing Pattern, *New Eng J Med* 273:1231, 1965.

DIFFERENTIAL DIAGNOSIS OF PULMONARY ALVEOLAR INFILTRATES

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Amer Rev Resp Dis 95(4):681–686, April 1967.

The pulmonary infiltrate is one of the most common problems faced by the physician. Yet, on reviewing the literature concerning the roentgenographic diagnosis of pulmonary disease, one quickly becomes confused. Infiltrates are described in photographic terms such as fluffy, hard, soft, nodular, linear, round, blurred, hazy, sharp, smooth, dense, et cetera. A more functional classification, one that can be appreciated roentgenographically, is to divide the infiltrates into those which primarily involve the alveolar spaces and those which primarily involve the interstitial structures.

The alveolar infiltrates can be recognized if they do one of three things: obliterate the vascular markings, form an air bronchogram, or fill an acinus of the lung forming a rosette pattern. This is in contrast to interstitial infiltrates which, by and large, accentuate the vascular pattern, thicken the alveolar walls giving a reticular or honeycombed appearance, or are lymphangitic in appearance. Most radiologists are well aware of the significance of the air bronchogram and of the blurred vascular markings. The filling of the acinus is not so well appreciated. When filled, it measures up to 0.5 cm. and has the appearance of a rosette. A more complete description of the rosette has been made by Ziskind. An exception to the above radiographic pattern is alveolar microlithiasis in its early stage. Although this is alveolar in nature, it fails to completely fill the acini and presents as small grains of calcium, which are in the subacinar structures. Fortunately, the density of the concretions makes the diagnosis relatively easy.

In Table 1 a classification is proposed using the type of material that may fill the alveoli. This combines the anatomic area of the lung involved with the type of pathologic material present. A further subclassification is undertaken in Table 2, showing the different disease states that may cause an accumulation of the various pathologic materials. It is beyond the scope of this paper to include these entities except in relationship to the roentgenologic and clinical characteristics that may allow their differential diagnosis, but pertinent references are given, which may allow the reader to study these entities in more detail. As can readily be seen, the number of entities in Table 2 is rather extensive. It would be even more extensive if all the bacteria, viruses, rickettsiae, and fungal agents that could cause alveolar disease in the lungs were listed. This is not really necessary, as the function of the radiologist in this case is to suggest the possibility of infection, and to show the extent of the pulmonary involvement and any complications present. The etiologic agent is determined, of course, by laboratory means. Chronic organizing pneumonia and sarcoidosis are listed separately as the etiologic agent under the entities causing pus in the alveoli, for these diseases have yet to be definitely classified. There is also a long list of disease states that may present with blood in the alveoli, and an even longer list of diseases presenting with edema in the alveoli, although this latter list is, of necessity, incomplete. The subdivisions of pulmonary edema are somewhat artificial, as the etiology of this entity still needs a great deal of clarification; but it is useful for clinical purposes to have such a classification. Despite the extensive number of diseases that may present with a pulmonary alveolar infiltrate, a rather perceptive diagnosis can often be made if the symptomatic

TABLE 1. *Pathologic Alveolar Materials* *

I. Pus
II. Blood
III. Edema
IV. Mucopolysaccharide
V. Fat
VI. Cells
VII. Calcium
VIII. Foreign material

* Modified from Ziskind and associates.

state of the patient is known, the effect on the pleura and hilar nodes are considered and, particularly, if the course of the disease is documented. Several of the entities, particularly alveolar microlithiasis and other metallic densities in the alveoli, can be diagnosed by their roentgenologic appearance alone.

TABLE 2. *Correlation of Infiltrates and Disease Entities*

- I. Pus
  - (a) Bacterial disease
  - (b) Viral disease
  - (c) Rickettsial disease
  - (d) Fungal disease
  - (e) Chronic organizing pneumonia
  - (f) Sarcoidosis
- II. Blood
  - (a) Infarction
  - (b) Trauma
  - (c) Idiopathic hemosiderosis
  - (d) Goodpasture's syndrome
  - (e) Periarteritis
  - (f) Uremia
  - (g) Blood dyscrasias
  - (h) Lymphoma
  - (i) Metastatic carcinoma
- III. Edema
  - (a) Increased capillary pressure
    - (1) Congestive heart failure; many etiologies
    - (2) Mitral stenosis
    - (3) Left atrial myxoma
    - (4) Cor triatriatum
    - (5) Pulmonary venous obstruction
    - (6) Overhydration
  - (b) Increased capillary permeability
    - (1) Hypoxic states
      - (a) Near drowning
      - (b) Incomplete pulmonary infarction
    - (2) Irritants
      - (a) Gases
      - (b) Liquids; aspiration of gastric acids
  - (c) Idiopathic edema
    - (1) High altitude
    - (2) Central nervous system disease
    - (3) Uremia
- IV. Mucopolysaccharide-alveolar proteinosis
- V. Fat
  - (a) Cholesterol pneumonia
  - (b) Lipoid pneumonia
- VI. Cells
  - (a) Bronchiolar cell carcinoma
  - (b) Loeffler's pneumonia
  - (c) Lymphoma and pseudolymphoma
  - (d) Metastatic carcinoma
  - (e) Desquamative interstitial pneumonia
- VII. Calcium-alveolar microlithiasis
- VIII. Foreign material
  - (a) Lipoidal
  - (b) Barium

TABLE 3. *Disease States Rarely Associated with Pleural Involvement*

- I. Pus
  - (a) Chronic organizing pneumonia
- II. Blood
  - (a) Idiopathic hemosiderosis
  - (b) Goodpasture's syndrome
- III. Edema
  - (a) Those associated with increased capillary permeability *
  - (b) Idiopathic group except uremia
- IV. Mucopolysaccharide—alveolar proteinosis
- V. Fat
  - (a) Cholesterol pneumonia
  - (b) Lipoid pneumonia
- VI. Cells
  - (a) Loeffler's pneumonia
  - (b) Pseudolymphoma
  - (c) Desquamative interstitial pneumonia

* Except incomplete pulmonary infarction.

If the patient is asymptomatic or only mildly symptomatic, consider sarcoidosis, alveolar proteinosis, Loeffler's pneumonia, or lipoid pneumonia. The most common causes of adenopathy that can be appreciated roentgenologically are sarcoidosis and lymphoma. Less frequent causes of adenopathy include idiopathic hemosiderosis, viral infection, fungal disease, and tuberculosis. If the pleura is involved, the entities listed in Table 3 can generally be excluded. After narrowing the possibilities by the previously mentioned methods, the subsequent course of the infiltrate may well give a definitive diagnosis. The relative rates of change for the various alveolar materials are listed in Table 4. It should be kept in mind that infiltrates may be mixed. For example, alveolar proteinosis may present with a superimposed pneumonia, or a patient in congestive failure may also have pneumonia or a pulmonary infarction. In the latter instance, the edema may clear promptly but an area of lung will show an alveolar infiltrate, which clears at a much slower rate. Many more examples can be given but, if the roentgenograms are carefully analyzed comparing clearing times in different parts of the lungs, the complications will become readily apparent.

The use of other clinical history that may be available is often helpful when looking at the initial roentgenograms, but be wary of compounding the clinician's error. For example, the clinical picture of pneumonia is often seen in idiopathic hemosiderosis and pulmonary infarction. Uremics may have an alveolar infiltrate, which is edema, blood, or pus.

TABLE 4. *Relative Rates of Change*

I.	Hours—edema
II.	Days—blood *
III.	Weeks—pus
IV.	Months—cells *
	Mucopolysaccharide
	Fat
V.	Years—calcium
	Foreign material

* Exceptions: Pulmonary infarction: changes in weeks; Loeffler's pneumonia: changes in days.

Alveolar infiltrates seen with cardiac enlargement and in a patient who is clinically in congestive failure may also be edema, blood, pus, or any of the materials listed in Table 1. It is only after the roentgenographic course of the infiltrate is plotted that one can become reasonably certain of the diagnosis.

Summary

The roentgenographic characteristics of alveolar infiltrates are the blurred vascular markings, the alveolar rosettes, and the air bronchogram. Although there is an extensive number of disease entities that may present this way, evaluation of the roentgenographic course of the infiltrate, along with the knowledge of the symptomatic state of the patient, the presence or absence of hilar adenopathy, or pleural involvement may allow one to suggest the probable diagnosis.

The writer wishes to thank Dr. Benjamin Felson for his review of the manuscript and helpful suggestions.

(The omitted figures and references may be seen in the original article.)

PROGNOSIS AFTER SURGICAL TREATMENT OF GRANULOMATOUS COLITIS*

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Boston, New Eng J Med 277(6):273–279, August 10, 1967. Reproduced with permission of The New England Journal of Medicine.

It has become clear that a number of patients with colonic inflammation, many of whom have previously been treated as having ulcerative colitis or one of its variants, actually have granulomatous colitis (Crohn's disease of the colon). Studies showing a significant correlation between microscopic granulomas in resected specimens of colon and the clinical and x-ray features described as characteristic of granulomatous colitis help to establish this as a valid clinical entity. The recognition of Crohn's disease in the colon and reports of recurrences after its operative treatment have led some to advise extreme caution in the surgical management of these patients, a view that seems to be shared by many. The recurrences have been primarily in patients with granulomatous ileocolitis, although some cases have been reported as well after operation for disease confined to the colon.

Our own observations led us to believe that patients with granulomatous disease of the colon, with or without ileal involvement, did well after total colectomy and ileostomy. In contrast recurrence was frequent in the few patients in whom intestinal continuity could be re-established by anastomosis. To obtain a systematic appraisal of this point we reviewed the entire experience in this hospital with operation for nonspecific inflammatory disease of the colon. The long-term results of operation for granulomatous colitis, both colectomy and ileostomy and less extensive procedures, have been compared with the results of total colectomy and ileostomy in ulcerative colitis, which is generally considered to be cured by this operation.

Methods

The pathological and diagnostic files of the New England Medical Center Hospitals were searched for all patients who had resections one or more years ago for inflammatory disease involving the colon.

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Patients whose clinical picture was clearly that of regional enteritis and in whom colonic involvement was only microscopic or immediately adjacent to the ileocecal valve were not considered to have granulomatous colitis.

The clinical histories were reviewed with particular attention to bleeding, fever, diarrhea, extraintestinal complications, proctologic findings, fistulas and distribution and types of lesions reported after barium enema and small-bowel x-ray examinations. The microscopical sections were reviewed by one of us without knowledge of the clinical histories.

Information on surviving patients not being closely followed by us was usually obtained by one of us from the patient by telephone. The relevant information was obtained from the patient's physician, a close relative and a prison infirmary in one case each. The patients were queried in sufficient detail to establish their current status and their courses since operation, with special attention to problems with the ileostomy and small-bowel function. In three cases in which it was suspected that a small-bowel lesion might be present, the patients had x-ray studies of the small intestine, a hemogram and assessment of the serum protein, prothrombin content and carotene.

For purposes of analysis, the patients were placed in three groups:

Proved granulomatous colitis. The sole criterion used was the presence in microscopical sections of one or more noncaseating granulomas containing giant cells, usually of the Langhans type, occurring in areas not adjacent to fistulas and abscesses. For the purpose of this study other pathological criteria of granulomatous inflammation were not accepted as sufficient for the diagnosis of granulomatous colitis since they depend to some extent upon individual interpretation.

Clinical granulomatous colitis. This group consisted of patients who had obvious clinical or x-ray characteristics of granulomatous colitis but who did not have granulomas in the surgical specimen. The clinical criteria included enteric or large and complicated perianal fistulas, absence of rectal involvement, segmental disease, extensive involvement of the small intestine and predominantly right-sided colitis, particularly with marked narrowing and thickening.

Ulcerative colitis. These patients had the typical clinical, roentgenologic and pathological picture of ulcerative colitis, without the clinical features described in the second group, and did not have granulomas in the surgical specimens.

In the analysis of the results patients who had had ileostomy and colectomy were separated from those with segmental resections of various types.

Results

General Remarks

Tables 1 to 3 present the results of this study. Forty-eight patients were considered to have ulcerative colitis, 21 to have proved granulomatous colitis, and 13 to have clinical granulomatous colitis. Follow-up study was possible in every patient but one (Case 1, Table 3). Extraintestinal complications were more frequent in Groups 1 and 2 as compared with the group with ulcerative colitis. These peripheral complications subsided completely in all patients whose bowel disease was controlled by operation. There were four cases of renal lithiasis in the follow-up period. One of the patients had a history of renal lithiasis before operation. Retarded healing of the perineal wound was of major significance in one patient (Case 3, Table 2), and was the ultimate cause of death.

Ulcerative Colitis (Table 1)

Forty-three of 48 patients survived operation, which was staged or unstaged total or subtotal colectomy and ileostomy in each. All 43 considered themselves well at the time of the follow-up study. The median follow-up period was six years, and the mean seven years, with a range of one to twenty-four years. There were two late revisions of ileostomies for stenosis at four and six years respectively. Both these patients, and 10 others in this group, did not have primary maturation of the ileostomy.* In neither was there any ileal disease initially, nor was there x-ray or pathological evidence of regional ileitis at the time of revision. One patient in this group reported some abdominal cramps and minor weight loss, and another, who had previously been grossly obese, had lost some weight. In still another patient episcleritis had recently developed, but there was no evidence of small-bowel disorder on x-ray examination or by chemical tests.

* Primary maturation of the ileostomy is a technical advance in the operative treatment of inflammatory colon disease in which the mucosa of the ileostomy is everted and sutured to the skin of the abdomen so that the serosal surface of the projecting ileum is covered with mucosa. Previously, the ileum was brought through the abdominal wall, and skin-mucosal approximation occurred by a natural eversion process over the course of a few weeks. This frequently led to functional and organic obstruction due to serositis and cicatrix formation.

TABLE 1. *Data in Patients Treated by Ileostomy and Total or Subtotal Colectomy.*

Type of Colitis	No. of Patients	Males	Females	Patients Surviving Operation	Deaths	Follow-up Period			Mean Duration before Ileostomy	Age at Onset		Extraintestinal Complications			
						mean	median	range		Range	Mean	total	arthritis	skin	other
						yr.	*yr.*	*yr.*	*yr.*	*yr.*	*yr.*	*no. of patients*	*no. of patients*	*no. of patients*	*no. of patients*
Ulcerative	48	28	20	43	5	7.0	6	1-24	4.9	10-80	33	11 (23%)	8	3	4
Proved granulomatous ‡	15	6	9	14	1	7.5	7	1-19	5.4	7-51	33	9 (60%)	7	4	1
Clinical granulomatous ¶	9	3	6	9	0	6.8	8	1-16	5.3	10-56	30	5 (56%)	3	2	2

Type of Colitis	Ileal Involvement				Type of Ileostomy		Revision		Current Status		Comment		
	total	minimal	moderate	marked	matured	not matured	post-operative	late	patient well	complications	retarded healing of perineal wound	renal calculi	loss of weight
	no. of patients	*no. of patients*	*no. of patients*	*no. of patients*	*no. of patients*	*no. of patients*	*no. of patients*	*no. of patients*	*no. of patients*		*no. of patients*	*no. of patients*	*no. of patients*
Ulcerative	11 (23%)	3	6	2	36	12	1,1*	2*†	43	1 patient each with minor dysfunction of ileostomy & wt. loss; wt. loss; episcleritis; & hilar lymphadenopathy.	6	2	2
Proved granulomatous ‡	8 (53%)	3	2	3	9	6	1*	4*§	11	1 patient with high output of ileostomy (no recurrent disease); & 1 with recent revision of ileostomy at 12 yr. & wt. loss (no recurrent disease).	5	2	4
Clinical granulomatous ¶	7 (78%)	3	1	3	4	5	3*	3*‖	7	1 patient (otherwise well) with asymptomatic pericholangitis; 1 late death (see text).			

* Original ileostomy not matured.
† At 4 & 6 yr.
‡ Granulomas in surgical specimen.
§ At 1-12 yr.
¶ No granulomas in surgical specimen.
‖ At 2-5 yr.

Proved Granulomatous Colitis, Treated by Ileostomy and Colectomy (Table 1)

Fourteen of the 15 patients survived operation and were living one to nineteen years later, with a mean follow-up period of seven and a half and a median of seven years. All patients considered themselves well at the time of follow-up study, with two exceptions: a patient whose disease had started as ileojejunitis and who required medical management of the persisting small-bowel disease after a total abdominoperineal colectomy performed four years previously had interrupted a rapid downhill course; and another whose stoma was recently revised because of a malfunctioning ileostomy. A total of four patients in this group had late revisions of the ileostomy. These four complications occurred among six patients in whom the original ileostomy was not matured primarily. In none was there x-ray or pathological evidence of regional enteritis at the time of revision. Four patients reported difficulty in maintaining weight, and another complained of abdominal cramps, for which no cause has been found.

Clinical Granulomatous Colitis, Treated by Ileostomy and Colectomy (Table 1)

There were nine patients in this group, all of whom survived operation. All patients except one, who died subsequently, and whose course is documented below, are currently well. The mean follow-up period in this group is six and eight-tenths years, the median eight years, and the range one to sixteen years. Two patients, in addition to the one who died, required late stomal revisions after four and five years. These three cases of dysfunction of the ileostomy occurred among five patients in whom the original ileostomy was not matured primarily. There was no pathological and no radiologic evidence of regional enteritis at the time of the revisions.

The history of the only patient in this entire study who died of *progressive small-bowel disease* after ileostomy and colectomy is as follows:

A 51-year-old woman was first admitted to the hospital in 1955 because of diarrhea of 2 years' duration. On the 2d admission in 1955 a diagnosis of "ulcerative colitis" was made because of radiologic changes mainly in the transverse colon and lesser ones in the ascending colon. The rectosigmoid was normal on x-ray study. Proctoscopy gave normal results except for two anal fistulas. She improved after treatment with corticosteroids. However, large necrotic lesions appeared on both legs and gangrene of the toes requiring amputations developed in spite of good peripheral pulses. The diarrhea increased and was followed by the development of a high-level rectovaginal fistula. Because of these factors, in November, 1957, a total abdominoperineal colectomy was performed. The ileostomy was not surgically everted. The colon showed punched-out ulcerations that were most striking in the transverse portion. The rectum and rectosigmoid were relatively normal, as was the ileum. Granulomas were found in the specimen only in the region of the large rectovaginal fistula.

Postoperative dysfunction of the ileostomy required reoperation on the 46th postoperative day, when a 21 cm. segment of the terminal small bowel was resected. No granulomas were found in this specimen, and the patient was discharged on the 40th day after the second operation.

Because of a retracting stoma there were cutaneous problems with the ileostomy, culminating in a deep ulcer adjacent to the ileostomy. In December, 1958, a Crile-Turnbull type of iloestomy was constructed, but because of necrosis, had to be revised on the 1st postoperative day. Three months later a large ulcer developed in the skin medial to the ileostomy. After many cutaneous problems with the ileostomy, episodes of fever, cramps and stomal obstruction, exploration was finally carried out in June 1959. The terminal 25 cm. of ileum and mesentery was thickened and congested, and was resected. Gross examination revealed deep ulcerations up to 2.1 cm. in diameter penetrating into the muscularis. No granulomas were found on microscopical examination except for one lymph node that contained a single, nonspecific granuloma. After operation a perforated duodenal ulcer developed and was closed, but the patient died in peripheral vascular collapse a few days later.

Pathological or Clinical Granulomatous Colitis Treated by Resection and Anastomosis or by Resection and Colostomy (Tables 2 and 3)

Of the 10 patients with and without granulomas in the surgical specimen who were treated by resections of various types, the gastrointestinal tract was reconstituted by anastomosis in seven. Four of these patients have had recurrences, in three of the four on two occasions. In each reoperation the recurrence was found to be primarily in the colon. The fourth patient with recurrence, which was suspected because of episodes of massive amounts of bright-red blood passed by rectum, refused x-ray examination.

In three patients with anastomoses there was no evidence of recurrence. However, one was lost to follow-up observation, a second died of perineal sepsis two years after operation, and the third is asymptomatic but has been receiving a small dose of corticosteroids, on the advice of her physician.

TABLE 2. *Data in Patients with Proved Granulomatous Colitis (Granulomas in the Surgical Specimen), Treated by Resection and Anastomosis or Partial Colectomy and Colostomy.*

Case No.	Sex	Age at Onset	Clinical Classification	Reason for Classification	Duration before 1st Operation	Extraintestinal Complications	Ileal Involvement
		yr.			*yr.*		
1	F	11	Granulomatous	Enterocutaneous fistula; right-sided involvement.	1	Arthritis	Minimal
2	F	18	Granulomatous	Antecedent regional enteritis	2	Erythema nodosum	Regional enteritis
3	M	13	Granulomatous	Ileocolic fistula & massive perirectal fistulas; initial right-sided involvement.	2	0	Regional enteritis
4	F	61	Granulomatous	Perianal fistula & stricture; segmental colitis.	6	0	0
5	M	25	Granulomatous	Segmental lesion	1/12	0	0
6	F	12	Granulomatous	Enterocutaneous fistula; right-sided colitis.	2	Erythema nodosum	Moderate

Case No.	Interval since Colectomy	Current Status	Comment
	yr.		
1	12	Clinically well (with recurrence)	Right-sided colectomy at age of 12; subtotal colectomy & ileoproctostomy for recurrence at age of 20; patient clinically well but protoscopic evidence of recurrence in colon.
2	6	Well	Jejunoileostomy bypassing all but 4½ feet of small intestine at age of 20 for regional enteritis with abscess; transverse colostomy for left-sided colitis & perianal fistulas (left-sided) proctocolectomy) at age of 24; patient well (overweight) at age of 30.
3	—	Dead	Resection of ileum & ascending colon, with ileotransverse colostomy & sigmoid colostomy, at age of 15; 3 mo. later, proctosigmoidectomy for perianal sepsis & fistula; 2 years later death after long course with nonhealing perineal wound & infection; patient never left hospital after first operation.
4	6	Well	Transverse colostomy at age of 67; left-sided proctocolectomy 3 yr. later.
5	6	Clinically well (with recurrence)	Segmental resection of hepatic flexure at age of 25; subtotal colectomy & ileodescending colostomy for recurrence at anastomosis 2 yr. later; radiologic & symptomatic evidence of recurrence at anastomosis; patient receiving salicylazosulfapyridine.
6	5	Well	Drainage of retrocolic abscess at age of 12; 4 mo. later right-sided ileocolectomy & ileotransverse colostomy; incision & drainage of retroperitoneal abscess at age 14; no fistula demonstrated; patient receiving small doses of steroids.

TABLE 3. *Data in Patients with Clinical Granulomatous Colitis (No Granulomas in the Surgical Specimen) Treated by Resection and Anastomosis and/or Partial Colectomy and Colostomy.*

Case No.	Sex	Age at Onset	Reason for Classification as Granulomatous	Duration before Colectomy	Extraintestinal Complications	Ileal Involvement	Interval since Colectomy	Current Status	Comment
		yr.		*yr.*			*yr.*		
1	M	58	Segmental involvement at hepatic flexure	1	0	None	—*	—*	
2	M	60	Left-sided segmental colitis	8	0	None	8	Dead	Patient died of cerebrovascular accident 8 yr. after left-sided proctocolectomy; no further bowel difficulty.
3	M	50	Stenosing ileocolitis	2	Arthritis	Marked	7	Living	Patient has had severe lower-gastrointestinal-tract bleeding on at least 2 occasions; persistent trouble with arthritis.
4	M	24	Segmental strictures of colon	1	Pyoderma; arthritis.	None	5	Living	Reoperation for recurrence in colon at 2½ yr.; patient now living with recurrence & asymptomatic after course of azathioprine.

* Patient lost to follow-up study, but seemed normal 6 mo. after operation at time of hernia repair.

Three patients had transverse colostomy and excision of the distal colon and the rectum. One died of a cerebrovascular accident eight years after operation, having had no further intestinal symptoms, and another was well six years after operation. In a third patient in this group left-sided colonic disease developed four years after a jejunoileostomy bypassing all but 4 feet of small bowel for regional enteritis with abscess. There has been no recurrence in the remaining colon six years after proctocolectomy and transverse colostomy.

Discussion

The prognoses of ulcerative colitis and regional enteritis after surgical treatment are quite different. In ulcerative colitis the patient is probably cured of his intestinal disease by operation, whereas in regional enteritis, recurrence is to be expected in at least 50 percent of the cases. If granulomatous colitis behaves like its counterpart in the small bowel, the avoidance of operation at all costs is justified since the patient might be subjected to the psychologic and physical handicap of an ileostomy only to face the disability of a recurrence later.

However, a careful review of the literature does not seem to support an overly pessimistic view of prognosis when total colectomy has been the treatment. Janowitz and his associates reported 12 recurrences after 30 operations for granulomatous ileocolitis, but it appears from a previous report that only three of the recurrences were in patients with ileostomy. The number of patients at risk after ileostomy cannot be gleaned from the data given. These authors reported no recurrences after operation in 10 patients whose disease was confined to the colon. Six of these patients had had total or subtotal colectomy and ileostomy, and the others resection and anastomosis. Lewin and Swales, in 48 patients operated upon for granulomatous colonic disease with minimal or no ileal involvement, found only one recurrence during a mean follow-up period of three years and eight months, and that recurrence was after anastomosis. The number of patients having total or subtotal colectomy and ileostomy in relation to the number having resection and anastomosis was not stated. Jones et al. reported one recurrence of "typical regional enteritis" proximal to an ileostomy thirteen years after operation in a group of 29 patients surviving total or subtotal colectomy and ileostomy for Crohn's disease of the colon. Some of these patients had ileal as well as colonic disease initially, although the patient with recurrence did not. These authors described 16 recurrences in 38 other patients in whom segmental resection or subtotal colectomy and anastomosis had been performed. The most discouraging experience is that of Brooke, who has reported two recurrences in the small intestine in four patients who had had total colectomy and ileostomy for granulomatous disease confined to the colon. He stated that this disease is not curable by surgical means. In the study of Colcock and his co-workers, even when the ileal involvement was extensive enough to be considered regional enteritis coexisting with "ulcerative colitis," the results of treatment of the colitis by ileostomy and colectomy were considered good in 17

of 20 patients. No recurrences were specifically mentioned in the three patients with other than good results. Thus, we have been able to find only six recurrences after ileostomy and colectomy for granulomatous colitis when the type of operation could be determined from the data given.

In the current study the ultimate results of treatment for both ulcerative and granulomatous colitis by ileostomy and colectomy have been excellent. In the only patient who died of progressively more proximal involvement of the small bowel after colectomy and ileostomy, it is difficult to separate mechanical or functional problems with the ileostomy from those related to the underlying pathologic process. Unlike the ileal recurrences of granulomatous disease described in the literature, this patient's trouble began immediately after operation—not after a symptom-free interval. Consequently, and because changes of typical regional enteritis were not seen on x-ray or pathological examination, the question of ileitis due to obstruction of the ileostomy should be raised. This complication was quite frequent before modern technics of performing ileostomies were introduced. We found no case of overt regional enteritis occurring proximal to an ileostomy stoma as described above. The paucity of such reports, corroborated by our own experience, suggest that this complication is the exception rather than the rule.

In the patients in this series whose disease was classified as granulomatous colitis and whose ileostomies were not matured primarily there were more late revisions of the ileostomy than in comparable patients with ulcerative colitis. However, late revision has not been required in either ulcerative or granulomatous colitis since primary eversion of the ileostomy has not been employed. Some of the patients with granulomatous disease in addition have had problems with maintaining weight, or with excessive output of the ileostomy and minor dysfunction, but this has not been due to demonstrable recurrent disease. An increased incidence of revisions of the ileostomy after treatment of granulomatous colitis has been reported by Hawk and Turnbull, who believed, however, that the small-bowel disorder was not due to stomal dysfunction.

Therefore, there is a problem in differential diagnosis in a patient with granulomatous colitis in whom small-bowel inflammation develops after ileostomy. The difficulty can be recurrent granulomatous inflammation but may well be due to a poorly functioning stoma. In our experience the problems of this type, with the exception of the fatal case presented in detail above, seem to have been solved by revision of the ileostomy. The more frequent dysfunction of the ileostomy in the patients with proved or suspected granulomatous colitis may well have been related in some unexplained way to the underlying pathologic process since the complication was strikingly less frequent in ulcerative colitis after comparable operations. However, if the correction of mechanical factors alleviates these problems, the method of management will be quite different from that required for recurrent granulomatous inflammation.

In this series there was a preponderance of patients with granulomatous colitis who had ileostomy and colectomy in contrast to some other reports on the same subject in which anastomotic procedures were in the majority. This does not represent a preference on our part for this operation over more conservative ones, but a choice that was dictated by the more or less total involvement of the colon in these patients or by destruction of the sphincters by fistulas. It is not clear why most of our cases have been of this type, but this has focused our main concern in granulomatous colitis on the possibility of recurrence after ileostomy.

In the seven patients in this series with sufficiently localized colonic disease to allow resection with anastomosis, there were recurrences in four patients, in three on two occasions. These recurrences were located on the colonic side of the anastomosis when the exact site could be documented. Despite this high recurrence rate after anastomotic procedures, the palliative value of operations that preserve sphincters must be considered before they are condemned.

This study and previous reports suggest that the patient who has granulomatous colitis or ileocolitis that can be resected by total colectomy and ileostomy has an excellent chance of long-term rehabilitation, if not cure, as a result of this operation. The results of this procedure for granulomatous colitis have not been markedly different from those for ulcerative colitis. In the granulomatous disease there should be some reservations about the permanency of the result although positive evidence of recurrent granulomatous disease in the ileostomy group was not found in this study irrespective of the presence or absence of ileal involvement at the time of operation. Therefore, a diagnosis of granulomatous colitis should not delay surgical treatment when conservative management fails to control the disease adequately.

Summary and Conclusions

To determine the prognosis of granulomatous colitis after surgical treatment, all cases of extirpative surgery for nonspecific inflammation of the colon at the New England Medical Center Hospitals were studied. Classification as *proved granulomatous colitis* required the presence of granulomas (21 patients); classification as *clinical granulomatous colitis* required the presence of typical clinical features of granulomatous colitis when granulomas were not found in the specimen (13 patients). Forty-eight patients were considered to have *ulcerative colitis*.

The prognosis in granulomatous colitis was not markedly different from that in ulcerative colitis when ileostomy and total colectomy was done. Recurrent disease was seen, however, in four of seven patients after resection and anastomosis for granulomatous colitis.

Dysfunction of the ileostomy was more frequent in patients with granulomatous colitis. These stomal problems occurred solely in ileostomies that had not been matured at the operating table. One death resulting from progressive small-bowel disease after colectomy and ileostomy for granulomatous colitis was attributed to this complication.

A diagnosis of granulomatous colitis should not delay surgical treatment when medical management fails to control the disease adequately.

(The references may be seen in the original article.)

HERPES SIMPLEX VIRUS AS A CAUSE OF ERYTHEMA MULTIFORME

Walter B. Shelley MD, JAMA 201(3):153–156, July 17, 1967.

Studies are reported on a patient with a seven-year history of recurrent attacks of erythema multiforme. In each instance, herpes simplex of the face preceded the generalized vesiculobullous eruption by seven to ten days. Intradermal skin tests with a formaldehyde inactivated herpes simplex antigen produced bullae which appeared to be erythema multiforme clinically and histologically. This is taken as evidence that erythema multiforme can be due to a hypersensitivity to the herpes simplex virus, and represents the first time erythema multiforme has been reproduced by an intradermal test.

Although erythema multiforme is a dramatic reaction pattern in the skin, its cause is elusive and goes unrecognized in most instances. In the patients in whom a cause may be suspected, drugs and internal malignancy have most often been indicted, but usually the relationship is tenuous and unsupported by laboratory data. The gravity of the clinical picture precludes drug challenges or reproduction of the complex of prior events. Nor can the disease be reproduced experimentally. Hence knowledge has lagged, and associated events (Table) have been cast in a sometimes dubious casual role.

In an attempt to shed new light on the problem, the present study concerned itself with a patient who gave a history of recurrent erythema multiforme, each attack being preceded by herpes simplex. Such a historical sequence, first noted by Urbach and repeatedly confirmed has remained enigmatic for over 30 years, despite the fact that it may be observed in more than 15 percent of those patients who have recurrent erythema multiforme. We asked whether these two disorders were indeed related and, if so, in what manner. In answer, we found that erythema multiforme developed in our patient as a unique hypersensitivity reaction to the herpes simplex antigen.

From the Department of Dermatology, Hospital of the University of Pennsylvania, Philadelphia.

Report of a Case

The patient, a 52-year-old white housewife, has a history of recurrent attacks of erythema multiforme for the past seven years. The onset was in February 1960 while the patient was on vacation on an island off the coast of Mexico. Initially diagnosed as a reaction to insect bite, vesiculobullous lesions continued to appear during the subsequent month while she was in Guatemala. They gradually subsided upon her return to the United States. Six months later a second attack occurred, notable for the severe oral ulceration which developed, again lasting about two months. The third attack was in the following spring and was related to the eating of large amounts

Attributable Causes of Erythema Multiforme

Drugs		*Contactants*
Arsenic	Mercurials	Bromofluorene
Barbiturates	Penicillins	Fire sponge (*Tedania ignis*)
Bromides	Phenolphthalein	Nitrobenzyl bromide
Digitalis	Salicylates	Poison ivy (3-pentadecylcatechol)
Gold salts	Sulfonamides	
Hydralazine hydrochloride	Tolbutamide	
Iodides	Trimethadione	

Infections	*Internal Malignancies*
Viral	Carcinoma
Herpes simplex, coxsackie virus, echovirus and other virus infections	Lymphoma
Mycoplasmal	Myeloma
Atypical pneumonia	Polycythemia
Bacterial	Radiotherapy thereof
Diphtheria, erysipeloid, glanders, pharyngitis, pneumonia, psittacosis, tuberculosis, tularemia, and typhoid fever	
Mycotic	
Coccidioidomycosis and histoplasmosis	
Protozoan	
Malaria, trichomoniasis, and vaginitis	

Collagen Diseases	*Vaccines*
Lupus erythematosus	BCG
Discoid	Poliomyelitis
Systemic	Smallpox
Dermatomyositis	

Food	*Hormonal Causes*
Margarine	Menses
Chocolate	Pregnancy

of chocolate. Subsequent attacks were erratic but in the last year she has had six distinct episodes.

It was early observed that each attack was preceded by herpes simplex of the lips, face, or nose. The appearance of herpes simplex was invariably followed seven to ten days later by a generalized attack of vesiculobullous lesions. Some iris and target lesions were observed, and the skin changes were most marked on the patient's legs. Erosions of the oral mucosa were common. A diagnosis of postherpetic erythema multiforme had been made by several of the dermatologists who had seen her.

Treatment had consisted largely of orally administered steroids. She had had trachoma and malaria in childhood, herpes zoster at age 30, hepatitis at age 41, and adenocarcinoma of the uterus at age 49. Therapy for the latter problem included panhysterectomy, bilateral salpingo-oophorectomy, and deep radiation. There was no evidence of recurrence. She had experienced generalized urticaria attributable to chloramphenicol (Chloromycetin). Her daughter had had a convulsive seizure following smallpox vaccination. The patient had been vaccinated with smallpox shortly before the first attack of erythema multiforme and subsequently without untoward developments.

Physical examination revealed round erythematous, slightly infiltrated lesions. These were widespread, but most common on the legs. Several bullae were noted and iris lesions were seen on the palm. A crusted herpetic lesion was observed on the lower lip. Otherwise, findings from the initial examination were unrevealing. Our studies survey a period of personal observation of more than a year.

Initial laboratory studies during hospitalization showed the following values were normal: hemoglobin, white blood cell count, fasting blood sugar, blood urea nitrogen, serum alkaline phosphatase, and serum protein electrophoresis. Results of the following tests of liver function were normal: serum glutamic oxaloacetic transaminase, serum glutamic pyruvic transaminase, lactic dehydrogenase, cephalin flocculation, and thymol turbidity. Stool cultures were negative for *Salmonella, Shigella, Staphylococcus,* and yeast. Intradermal skin tests to the following showed no hypersensitivity at 20 minutes and 48 hours: trichophytin, *Staphylococcus aureus,* both orange and white forms; *Neisseria catarrhalis;* viridans type *Streptococcus;* hemolytic *Streptococcus;* anhemolytic *Streptococcus; Strep faecalis; Aspergillus; Penicillium; Botrytis cinerea; Monilia sitophila; Mycogone nigra; Escherichia coli; Proteus* X; *Corynebacterium pseudodiphtheriticum;* purified protein derivative (PPD), 0.001 mg; histoplasmin; coccidioidin; blastomycin; *Phycomycetes; Fusarium vasinfectum;* Dermatiaceae; *Paecilomyces varioti;* and *Chaetomium.*

Smallpox vaccination induced a normal local immune response, and had no effect on the erythema multiforme. The absolute basophil count was 20/cu mm. Direct basophil degranulation tests were negative for antibodies to egg albumin and gluten. X-ray films of the chest and sinuses, and serial x-ray films of the upper and lower gastrointestinal tract revealed no abnormalities.

Findings from proctoscopic and gynecologic examinations were normal, except for the absence of the uterus from hysterectomy. A Papanicolaou smear was class I.

Special virologic studies were done.

1. Virus was isolated from a typical 48-hour old herpes simplex lesion of the nose prior to the attack of erythema multiforme with the following results (human embryo kidney tissue culture): vesicle swab, + +; vesicle fluid, +; and saliva, −.

2. Neutralizing antibody titer was as follows: total N (neutralizing antibodies), 1:32; IgG, 1:32; IgA, trace to none; and IgM, trace to none.

3. Study of precipitating antibodies (gel diffusion) revealed a strong precipitin band against one component of the herpes simplex virus antigens.

4. Study of immunoglobulins showed IgG level was normal, IgA level was above normal, and IgM (two components) level was normal.

Smears of the base of the erythema multiforme bullae revealed no giant or balloon cells. A biopsy of one of the lesions disclosed an epidermal bulla, edema, angiitis, and lymphocytic infiltration. No giant cells, eosinophils, or organisms were present. Special stains (PAS, Fite-Faraco, and Giemsa) showed nothing additional. No viral inclusion bodies were seen.

Special studies were made with a herpes simplex antigen. (The vaccine was prepared by growing the herpes simplex virus on rabbit kidney, followed by formaldehyde neutralization. Thimerosal [Merthiolate] was used as a preservative.) Intradermal injection of this vaccine (0.01 ml) produced a severe 1.5 cm local bullous reaction at 48 hours which duplicated the clinical lesion—even to the fact that the vaccine induced bulla showed the most intense activity at the borders as in a target lesion. This was repeated three times with an identical bullous reaction developing which on biopsy was histologically the same as the clinically evolving lesions. Not only was a local reaction triggered, but 48 hours after the skin test distinct vesiculobullous lesions appeared elsewhere in the skin. Healing was followed by prolonged postinflammatory pigmentation. Dilution of the vaccine (1/20) produced the same local erythema multiforme reaction. Significantly, intradermal injection of the diluent, ie, tissue culture medium 199, edetic acid (Versene) and thimerosal, as well as poliomyelitis vaccine (Salk) (containing formaldehyde, monkey kidney, and poliomyelitis viral antigen) elicited no local or distant responses. Observations on 15 other patients who had a history of uncomplicated recurrent herpes simplex showed no vesicular or bullous reaction to the herpes vaccine despite administration in a dosage 100 times as great as that which was employed in the skin tests with this patient.

Comment

The intriguing relationship of recurrent herpes simplex and erythema multiforme has been most carefully studied and reviewed by Nasemann. In a large series he found that 15 percent of all cases of erythema multiforme were regularly preceded by an attack of herpes simplex. Serologic tests, including neutralizing complement fixation, agglutination, and precipitin gel diffusion, disclosed nothing unique to these individuals.

Little has been reported concerning herpes simplex virus as a possible antigen. It is well known that inactivated herpes simplex virus may at times produce a delayed papular tuberculin type response on intradermal testing of patients with recurrent herpes simplex. In several patients with postherpetic erythema multiforme, skin testing by previous observers revealed nothing unusual, but Söltz-Szöts did report a case in which erythema multiforme developed while the patient was receiving a herpes simplex vaccine for the treatment of recurrent herpes simplex.

In our present study we have repeatedly reproduced the local vesiculobullous lesion of erythema multiforme by the intradermal injection of a commercially prepared but as yet experimental herpes simplex antigen. This vaccine has been previously used by other investigators and found to be ineffective in the therapy of recurrent herpes simplex. No vesicular or bullous reactions were noted by them and in our control group of 15 the typical delayed papular response only was seen. Thus the vesicular reaction is viewed as evidence of a specific type or degree of hypersensitivity limited to patients with postherpetic erythema multiforme. It should be stated that the vesiculobullous reactions were limited to our postherpetic erythema multiforme patient, and were not seen in two other patients with erythema multiforme. In the latter two patients, sulfonamide sensitivity and adenocarcinoma of the sigmoid colon were found to be causative. They had no history of having had herpes simplex.

On the basis of the limited experience recorded here, it is recommended that a herpes antigen be made available for skin testing patients with erythema multiforme to ascertain which may have erythema multiforme due to a specific hypersensitivity to the herpes virus. The *vesicular* skin test may offer a way to objectively distinguish postherpetic erythema multiforme from the other forms. It is important to realize that herpetic erythema multiforme may be more common than we suspect from the history since many of the imputed causes may simply serve to trigger inapparent or hidden herpes simplex. Thus menses or pregnancy might activate herpes simplex of the vaginal or vulvar area, and in turn cause erythema multiforme in an appreciable number of patients; recent studies reveal that nearly one in

every 500 Papanicolaou smear tests show intranuclear inclusions suggestive of herpes simplex virus infection.

The oropharynx, conjunctiva, penis, or indeed the central nervous system may be other occult reservoirs of the virus which may be activated by a welter of diverse diseases (Table) and thereby induce erythema multiforme. It is evident that only a mass skin test screening of erythema multiforme patients will permit an estimate of actual role played by herpes in the production of this disease. It should be stressed apparently that one must employ a potent standardized antigen identical or similar to that used by us. It is probable that the crude weak antigens used many years ago led to the negative results previously reported. Finally, erythema multiforme is not the only postherpetic syndrome which has been described. Shearer and Finch recently reported a severe periodic organic psychosis appearing 2 to 11 days after each attack of herpes simplex. It would appear valuable to explore by skin testing this and other avenues of disease in which herpes appears to have a temporal relationship. Hence, the herpes antigen which so far has had indifferent success as a therapeutic vaccine may find a more distinguished career as a diagnostic agent.

Tokumaru's studies would indicate that this patient had recurrent attacks of herpes simplex as a result of an inability to form adequate amounts of herpes specific IgA immunoglobulin. Much remains to be done to delineate the immunologic parameters of this bullous vasculitis pattern seen when the viral antigen was introduced into the skin. Possibly the elevated nonspecific IgA immunoglobulin levels found are in some way related. Certainly the fact that inactivated herpes virus can reproduce the disease locally gives us an experimental clinical mode for further study, and provides hope that specific tumor and drug antigens may be prepared for this type of skin testing.

This investigation was supported in part by a grant from the John A. Hartford Foundation.

Paul Gross, MD, referred the patient. Tadasu Tokumaru, MD, did the virologic studies.

The herpes simplex antigen used in this investigation was supplied through F. B. Peck, Jr., MD, of the Lilly Laboratory for Clinical Research, Indianapolis.

Generic and Trade Names of Drugs

Chloramphenicol—*Chloromycetin.*
Thimerosal—*Merthiolate.*
Hydralazine hydrochloride—*Apresoline Hydrochloride.*
Tolbutamide—*Orinase.*
Trimethadione—*Tridione.*

(The omitted figures and references may be seen in the original article.)

MEDICAL ABSTRACTS

INCREASED ELASTIC TISSUE IN THE DERMIS: THE DERMAL ELASTOSES

J. Graham Smith, Jr., Bull NY Acad Med 43(3): 173–185, Mar 1967.

Elastic tissue is different morphologically, physically, and chemically from collagen and, although it is invariably associated with collagen, is not derived from collagen.

Emphasis is placed on dermal disorders associated with increased elastic tissue to draw attention to these fibers, which also play an important role in connective tissue disease.

Elastic tissue stains appear to be quite specific and, in those elastoses studied extensively by other techniques, abnormal increases in elastic tissue have been confirmed. There is no evidence as yet to suggest that the material that stains with elastic tissue stains represents a degraded or abnormal collagen.

Experimental methods available for production of increased elastic tissue in the dermis such as ultraviolet rays and chronic acidosis should aid the study of the synthesis of this fibrous protein.

LAPAROTOMY FOR JAUNDICE

J. B. Bourke MA MD Cantab, P. Cannon MA MB Cantab MRCP, H.D. Ritchie MA Glasg ChM Edin FRCSE FRCS, Lancet II (7515):521–523, Sept 9, 1967.

One hundred fifty-five jaundiced patients underwent laparotomy at the London Hospital between

1961 and 1965. In three, the cause of the jaundice turned out to be medical rather than surgical. There was no mortality. The steroid diagnostic test, percutaneous liver biopsy, and percutaneous transhepatic colangiography produced misleading or unhelpful results in some cases, and serious complications in others. If the cause of the jaundice is in doubt, observation of the patient should continue for not less than three weeks, by which time the diagnosis will often have become apparent. If a diagnosis has not been made after six weeks of jaundice, laparotomy should be carried out.

THE AMINOACIDURIAS

Mary L. Efron MD and Mary G. Ampola MD, Pediatric Clin N Amer 14(4):881–903, Nov 1967.

The primary aminoacidopathies were last reviewed in the Pediatric Clinics of North America in 1963. Since that time, mass screening for phenylketonuria and rapid expansion of the programs to include galactosemia, maple syrup urine disease and many other inborn errors of metabolism, have focused attention on these rare diseases. Ten years ago the study of biochemical causes of neurological disease was an esoteric corner of medicine; today most patients referred because of mental retardation or other neurological findings are tested for biochemical defects as a routine.

LATE SYPHILIS—A REVIEW OF SOME OF THE RECENT LITERATURE

Leslie Nicholas MD and Herman Beerman MD DSc (Med), Amer J Med Sci 254(4):549–569, Oct 1967.

The general recent increase of syphilis morbidity, in spite of modern therapy, will undoubtedly lead to development of various late lesions. The present review is aimed at covering some of the literature published for the most part in the last 5 years, pertaining to this phase of syphilis.

In the attempt to arrive at a diagnosis, or at least a working diagnosis, there is a great tendency today to skim over the history, to neglect the fine points of physical diagnosis, and to depend almost entirely on the laboratory reports. If this be the case, it is most important for the clinician to request the proper laboratory procedures. There has been a tendency lately on the part of many physicians to omit a routine serological test for syphilis (STS). In fact, in many hospitals where an STS was formerly performed on each patient admitted, the blood serum now is so tested in as few as 11.9%. No laboratory report can ever replace a good history or physical examination, or both, but it can arouse the suspicions of the examiner and a high index of suspicion is often the motivating force needed to arrive at a diagnosis of syphilis.

MAN VERSUS ARTHROPODS

Cyril H. March MD and Alexander A. Fisher MD, GP 36(4):115–121, Oct 1967.

Arthropods (insects and arachnids) affect man by provoking toxic, allergic or mixed reactions. The local and systemic toxic reaction is exemplified by the bite of the brown recluse spider. Large doses of corticosteroids are helpful in many of these syndromes. Intralesional steroids may be beneficial in papular urticaria produced by fleas and bedbugs. Eradication of caterpillar dermatitis requires washing of the clothing and bedding contaminated by the venomous caterpillar hair. At present, topical repellents offer the best protection.

DISEASES OF MUSCLES—CLINICAL MANIFESTATIONS AND DIFFERENTIAL DIAGNOSIS

Louis D. Boshes MD, New Physician 16(10):263–274, Oct 1967.

Disorders of muscle may be caused by trauma, infection, inflammation, congenital defects, primary degenerations, metabolic or nutritional defects, endocrine diseases, tumors, toxic states, circulatory or vascular lesions, and disorders of excitability such as myasthenia gravis. Diagnosis is determined by the physical findings, the clinical history, pharmacological tests, and pathologic changes noted on muscle biopsy. A classification is also given.

DENTAL SECTION

PERIODONTAL CONSIDERATIONS IN THE CONTOUR OF RESTORATIONS

Sheldon D. Benjamin DMD, Reprinted by special permission from the Journal of Southern California Dental Association, Vol. 35, pp. 254.

There exists in dentistry an intimate relationship between the quality and type of restorative dentistry and periodontal health. One important factor that is often overlooked is the contour of restorations. Recently a meeting of the Operative Dentistry Division of the University of Southern California School of Dentistry was held concerning this subject. The objective of the meeting was to develop a unified concept regarding the contour of restorations that could be presented to students. Some of the concepts that were presented are:

1. *Tooth Morphology*. A need exists for review of basic tooth morphology by both the student and the dentist.

2. *Oral Hygiene*. It has been amply demonstrated in the literature that the greatest single etiologic agent in the creation of periodontal disease is dental plaque. Most of our concern with tooth contour is to either prevent the accumulation of plaque, or if it does accumulate to provide accessibility for its removal. Overcontouring of restoration, particularly at the cervical portion, is undesirable since this leads to plaque accumulation at the vulnerable gingival margin area.

3. *Embrasure Design*. Sufficient embrasure space must be provided in all areas, with the gingival embrasure being of prime importance. There must be adequate room for the gingival papilla. Access must be provided for cleansing. The occlusal embrasure is important in the prevention of food impaction. The occlusal embrasure should be the depth of a strand of dental floss. Buccal and lingual embrasures must be sufficiently pronounced to permit the cleansing action of the cheeks and tongue.

4. *Partial vs Full Coverage*. Partial coverage in a periodontally prone patient is generally superior to full coverage when possible. This leaves portions of the natural tooth for orientation during fabrication of the restoration. Less subgingival marginal area is present with partial coverage which is significant in a periodontal patient. Another problem in full coverage is that many operators do not sufficiently reduce the gingival portion of the preparations, particularly in porcelain fused to gold or veneered crown preparations. This leads to an overcontouring at the critical gingival area with compression of the tissue and plaque accumulations.

This is certainly a perfunctory glance at a complex problem. It is hoped that the practitioner will become more aware of the importance of crown contour in his practice and encourage some debate and clinical studies in this area.

BACTERIAL ENDOCARDITIS AFTER DENTAL PROCEDURES IN PATIENTS WITH AORTIC VALVE PROSTHESES

H. O. Archard and C. W. Roberts, JADA 72(3): 648–652, Mar 1966.

As technics in open heart surgery advance, the likelihood of a patient with a prosthetic valvular replacement seeking routine dental treatment increases.

That we should be cognizant of the problems associated with such treatment is described in the above article.

The use of prophylactic antibiotics for patients who have rheumatic or congenital heart disease is well established. It is the authors' contention that this is inadequate coverage for patients with prosthetic valves and cites two cases of bacterial endocarditis developing in patients who had received Procaine Penicillin, 600,000 units I.M., B.I.D. before and after the dental procedure. Both patients died.

The regimen currently employed at National Heart Institute, National Institute of Health, Bethesda, Maryland, is:

1. Procaine penicillin—600,000 units intramuscularly every six hours the day before, day of, and three to five days after dental procedure.

2. Streptomycin—0.5 Gm. intramuscularly every 12 hours, the day before, day of, and for three to five days after dental procedure.

3. Aqueous penicillin—1,000,000 units intramuscularly one hour before dental procedure.

4. Methicillin—4 Gm. intramuscularly or intravenously every six hours, day of, and three to five days after dental procedure.

5. Broad spectrum antibiotic troches on each of two days before dental procedure.

(Abstracted by: CAPT Howard S. Kramer, Jr., DC USN.)

Holiday Season Greetings

As the Christmas Season arrives again with its religious connotations, hopes for reunions, gratefulness for our way of life and, for some, days of relaxation, let us be thankful as a Corps.

During the times of devotion let us pray for our comrades who are away from their families at duty stations overseas. Throughout the holiday season let us extend greetings and good cheer to their families who are here in the United States.

Merry Christmas to all officers, enlisted men, civilians, and their families. May your blessings be many!

Frank Kyes

F. M. KYES
Rear Admiral, DC, USN
Assistant Chief of the Bureau of Medicine and Surgery (Dentistry) and Chief, Dental Division

MALTESE—U.S. NAVY DENTAL SEMINAR

The first Maltese-American Navy Dental Seminar was held aboard the destroyer tender USS YELLOWSTONE in Valletta, Malta, on October 7, 1967, by the Dental Department of the USS YELLOWSTONE, the Dental School of the University of Malta, and Dental Association of Malta to exchange ideas and information in the field of dentistry between Maltese and American Navy dentists.

The Dental Seminar was opened by the head of the YELLOWSTONE Dental Department, CDR H. E. Freeburn, DC USN, and the Maltese guests were officially welcomed aboard by the Commanding Officer of the YELLOWSTONE, CAPT W. M. Cone, USN. Professor J. J. Mangion, the Dean of the Dental School of the University of Malta, introduced the Maltese speakers, Doctor C. Olivieri Munroe, who spoke on "Oral Disease in Malta: Epidemiological Aspects," and Doctor G. E. Cammilleri, who spoke on the "Diagnostic Value of Exfoliative Cytology." The American speakers were LCDR Carl J. Smith, DC USN, and LT Harold J. Murray, DC USNR. Doctor Smith lectured on "Application of a Technique for Pin-Reinforced Amalgam," and Doctor Murray spoke on, "A Technique for Immediate Root Resection." Dental students from the University and members of the Dental Association of Malta attended the meeting.

DENTAL CLASSIFICATION OF RECRUITS

In a survey conducted on 1,595 recruits during the period 22 June–3 July 1967 at Naval Training Center, Great Lakes, Illinois, the following statistics afford an indication of the dental classification of recruits at the beginning and upon completion of their nine-week recruit training.

Classification	*Number of Recruits Upon Induction*	*Number of Recruits Upon Completion*
I	268–16.8%	412–25.8%
II	861–54.0%	715–44.8%
III	388–24.3%	307–19.3%
IV	60– 3.8%	33– 2%
V	18– 1.1%	0– 0%

At the end of the training period, 128 records or 8.1% were not available for classification because of reassignment to other companies or activities, or separation from the naval service.

NURSE CORPS SECTION

NURSES NOTES FROM THE USS REPOSE (AH–16)

The following excerpts have been taken from communiques received from the USS REPOSE:

"Much as happened to the 'Angel of the Orient' during the past year. Change of Command Ceremonies were held in November 1966 when the ship's Commanding Officer, CAPT E. Maher was relieved by CAPT Ray Gossom and again in September 1967 when CAPT Rudolph Nadbath was relieved as Commanding Officer of the hospital by CAPT Herbert Markowitz. April 1967 marked

a sad farewell to our Chief Nurse, CDR Mary Kovacevich, NC USN, who went to Great Lakes Corps School and we welcomed her relief, CDR Lorraine Hankey, NC USN, from NH Pensacola, Florida. At the crack of dawn on the 10th of April, we sighted our sister ship, the USS SANCTUARY, as she entered DaNang Harbor for the first time. AH–17 was indeed a pretty sight and many of us were able to tour her and visit with old acquaintances before we set sail for Subic, after forty-nine days 'on station'.

"Travelwise our 'ashore time has been spent at beloved Subic Bay for routine maintenance and repairs. Although not an R and R port, land is land, and we have enjoyed our seven to ten day breathers in Manila and beautiful Baguio City. We did visit Hong Kong from 26 December to 2 January and the Hilton Hotel, with its room service and wonderful bathtubs, sightseeing tours, shopping the Walla Walla boats, candlelight dinners, dancing and a glorious New Years Eve won't soon be forgotten. Bangkok, Japan, Taiwan and a return to Hong Kong were tentatively scheduled but operational commitments forced their cancellations. Singapore, however, was a reality. We arrived there on the 7th of August and berthed alongside the HMS BULWARK and the HMS FEARLESS at the HMS Terror Naval Base. The Royal Navy proved to be more than a perfect host. Several cocktail parties, club luncheons, ship's dinners with authentic Happy Hour refreshments and a boat picnic were accorded us. This, plus the shopping and 'bargaining' with the local merchants for exotic purchases, the colorful taxi tours, the gourmet delicacies, the phone calls to loved ones at home and, of course, the hotel accommodations, complete with bathtubs, made the next four days a delightful memory.

"Another highlight of our Singapore visit was our crossing of the Equator on August 6th. All REPOSE 'pollywogs' were duly initiated into the Solemn Mysteries of the Ancient Order of the Deep and became worthy, trusted and envied 'shellbacks' a truly gory, slimy, crazy and all around fun event. There are now 'shellbacks' in the Navy Nurse Corps.

"Ship life is confining and yes, even routine to some, however, the call 'flight quarters' soon relieves the monotony. Our newspapers are usually a week or so outdated, our coke supply can dwindle and it seems our mail can take forever and a day to reach us. Woe is us you say? No siree! ! Guaranteed work takes up many an hour but we do have free time. Song fests, birthday parties, hail and farewell shindigs, tape sessions, visiting celebrities—Martha Ray, Hugh O'Brien, Raymond Burr, Robert Stack; General Greene, Commandant of the Marine Corps, General Walt, Commanding Officer Marine Amphibious Forces—Vietnam, and his replacement, General Cushman; Admiral Brown, the Surgeon General of the Navy; our Ambassador to Vietnam, Mr. Ellsworth Bunker, General of the Army, Omar Bradley, Admiral Ward when he presented the Navy Commendation Award to the USS REPOSE, our 'on camera' appearance when CBS, NBC and movieland people were filming our 'home away from home,' the St. Patty's Day celebration, the off broadway spectaculars, 'Fantail Follies' number one and number two in March and July, the Navy Nurse Corps Anniversary, the beach parties at Repose Beach in DaNang, the Sunday brunch, the steak fries, the Saturday evening buffet, the bridge tournaments, the showing of home movies, the CARE packages from CONUS and the radio shack 'phone patches' are but some of our log entries.

"However, it isn't all fun and games. We work and we would like to think, work well. Our doctors are just superb and the Lord knows their task isn't an easy one. Likewise our nurses. Crowded ward conditions—peak census of six hundred forty-seven patients in May—do not hamper their unheralded performance. Our corpsmen do not have to take a back seat to anyone, as evidenced by the fact that the Operating Room was able to effectively run three rooms during March, when three hundred ninety-one procedures were performed and again in May when our figures reached four hundred fourteen. This was accomplished by six technicians and two inexperienced on the job trainees. Needless to say, many long, sleepless and hungry hours were well spent. This, together with the team performance of personnel in the Recovery Room, Intensive Care Unit, Central Supply, Blood Bank and Lab, plus the not to be forgotten outstanding ward personnel, makes us proud members of AH–16, humorously known in WesPac as Attack Hospital 16; since we 'kissed' an oiler during a refueling operation on June 12 requiring us to go on the serious list and necessitated a 'dry dock recuperation period' in Subic Bay.

"All in all, we have indeed been fortunate. Our patient turnover has greatly increased but our mortalities have remained at a low level. Our many 'chopper touchdowns' have been accident free thanks to the skill of the pilots and the excellence

of our ship's line officers. Our supplies can reach an all time low but as of now we've managed to have the right things at the right time. Our morale can falter now and then, somehow forty to fifty-five days at sea can get the best of you, but one glimpse of the battle torn young men arriving at Triage banishes all of our discontentments.

"In conclusion our performance since January 1967 to us is commendable. We have admitted four thousand eight hundred and thirteen patients to our wards, accomplished one thousand nine hundred forty-seven surgical procedures including ten open heart operations and one portal caval shunt, landed four thousand six hundred sixty-five choppers on our flight deck without incident. On 29 July our ship was underway within twenty-eight minutes enroute to the Gulf of Tonkin to render assistance to the USS FORRESTAL, which was burning and had many seriously injured casualties on board.

"Yes, it has been a good ten months. We have all gained professionally from our experience. Most of us have more insight, are more tolerant, more understanding and all should leave our beloved 'white bucket' better people. We might add with more sturdy sea legs too, especially after our three day encounter with Typhoon Nancy and the other squalls that cross our bow."

AEROSPACE MEDICINE SECTION

MASS CASUALTY HANDLING ABOARD CARRIER — PART I

The following is a medical account of the disaster aboard the USS FORRESTAL (CVA–59), on 29 July 1967, based on a letter from CDR L. A. Herrmann, MC USN, Medical Officer.

In the Tonkin Gulf on the morning of 29 July, we had completed sick call and by 1000 all was quiet in the Medical Department. At approximately 1050 H, "Fire on the Flight Deck" was announced over the 1MC. Within a couple of minutes "General Quarters" was called away and almost simultaneously there was a series of explosions which rocked the ship. I, along with the surgeon, LCDR G. G. Kirchner, reported to the Main Battle Dressing Station. We had no idea at that time what had happened, but judging by the explosions, the like of which I had never experienced, I felt that things were pretty serious.

Within minutes the first four casualties had arrived. These were massive burns and shrapnel wounds, one requiring an immediate tracheotomy. It became evident that we should switch into our mass casualty plan. Incoming cases were routed to Ward II for triage and emergency treatment. The treatment room and physical examination room (Aviation Medicine) were set up for treatment and suturing, and manned by corpsmen. There were approximately 75 casualties who received definitive care, including tetanus booster, and prophylactic penicillin 600,000 units and Streptomycin 0.5 Gm I.M. at these facilities and returned to duty.

Fifteen patients were in the sick bay at the time of the disaster. All but two of these were evacuated to the medical office or returned to duty. Within an hour every bed was filled and overflow of casualties were kept on litters. As Doctor Kirchner continued directing triage and emergency medical treatment, I circulated through the department supervising the overall operation, assisting as needed with emergency care, and directing the flow of supplies, etc. I learned that the assigned teams for the After Auxiliary Battle Dressing Station (BDS) 03-202-1-L and After Battle Dressing Station 2-222-2-L were unable to get to their stations and were therefore put to work in sick bay. That gave us one additional dental and medical officer. All casualties (including burns with wounds) received antibiotic and tetanus prophylaxis. I. V. fluids (Ringer's Lactate) were started on about one-half of the casualties and Morphine (15 mg) was administered as needed.

Within the first hour it became evident that we could use additional medical assistance. Knowing the Oriskany was nearby, I had the bridge notified to request assistance. Very rapidly two medical officers, one dental officer, and 13 corpsmen arrived via helicopter. The two medical officers were taken to the forecastle to assist one of our dental officers who was using this as a holding area for his Forward Auxiliary Battle Dressing Station. By the time this medical augmentation arrived, primary triage and immediate emergency medical care had been

accomplished. Patients were then selected for evacuation to the two other carriers. At about 1200 H, 10 ambulatory, treated wounded were transferred to the flight deck for evacuation to Oriskany. At about 1230 H, five treated, litter patients were sent to the flight deck for helo evacuation to Bon Homme Richard. These were followed by five more litter cases at 1300 and three more at about 1330. Ultimately, 33 cases went to Oriskany and six cases to Bon Homme Richard. This reduced our patient load to a number we could definitively handle. Ultimate casualty count numbered one hundred thirty-four killed, 90 admitted for treatment, and 75 treated and returned to duty.

We were also joined by LT George Hamilton, MC USNR, from USS Rupertus and Surgeon LT Leo Barnett from HMAS Hobart. They had arrived within that first hectic hour and immediately started working on the wards. The Medical Officer of Oriskany, LCDR A. Adeeb arrived. Since the situation seemed stable and under control, and in view of the evacuation to the other carriers, it was felt that Doctor Adeeb should return to his ship. (Ed. Note: Part II will describe the experiences aboard Oriskany.) Later in the afternoon we returned the other Oriskany physicians. Two doctors who had arrived from the Bon Homme Richard were almost immediately returned to their ship to assist with casualty handling there.

Minor injuries continued to flow through the treatment areas in sick bay throughout the afternoon, including numerous cases of smoke inhalation. One of the latter was dead on arrival, one died after about two hours, and others were retained in sick bay for approximately 24 hours.

At about 1530 H Doctor Kirchner and I went to the forecastle to survey the patients held there. Cots had been kept available there for transient personnel, and these were now set up and utilized for patients. The forecastle is roomy, well lighted and makes an excellent casualty holding station. Its only drawback is difficult access for litter patients. Three casualties had been evacuated from this area to the other carriers. With the exception of these three patients, all remaining casualties (approximately 12), were then evacuated to sick bay.

Throughout the early phases of the disaster the three BDS's which we were able to man (Forward Auxiliary, Midship Auxiliary and Forward BDS) functioned effectively. Supplies in the BDS's were adequate except for injectable analgesics.

By late afternoon we had begun to accumulate many deceased. A morgue was established in Hangar Bay One. A team of corpsmen and a dental officer were assigned there to tag, identify and finger print the bodies. I anticipated that when we rendezvoused with Respose she would be able to take the deceased, as we had three days until arrival in port and insufficient facilities aboard. Doctor Kirchner and I started definitive surgery in the operating room at about 1630 H. Our two flight surgeons and the two doctors from the destroyers continued on the wards, seeing to the continuing supportive care of the burns and smoke inhalation victims. Charts were made up on all patients and routine patient care and charting initiated. At dusk Doctor Barnett was returned to HMAS Hobart, and at day break Doctor Hamilton was returned to USS Rupertus. By 0400 H we secured one flight surgeon and by 0600 H the second. We continued in surgery until 0600 H, 30 July.

At about 2230 H the ship rendezvoused with Repose and transferred seventy-seven deceased and six patients. We also transferred one Dental Officer with dental records of the missing to assist in the identification of the dead. (Ed. Note: Repose also received additional casualties from the other carriers.)

Throughout the disaster we were fortunate to have access to both of our storerooms, and necessary medical supplies were adequate except for Kerlex dressing and Ringer's Lactate. Additional supplies of these items were obtained from the other carriers.

Through the evening and the next day attempts to recover additional dead from burned-out areas were hampered by dense smoke, flash fires and chlorine gas. On the following day, however, these areas were slowly cleared and explored, and additional remains recovered. Almost all of these bodies were carbonized and many were fragmented. Recovery of bodies continued on through the second and third days, the last being recovered in the afternoon of 31 July. The dental department worked tirelessly and under the most adverse conditions in assisting us in identification of the bodies.

Musters of the crews were held on 30 July and 31 July, but were not judged complete, thus impairing the accurate listing of the dead and missing. On arrival in Subic Bay, 31 July, an aircraft was waiting adjacent to the pier to transport the remaining fifty-two bodies to the mortuary at Clark Air Force Base.

The Commanding Officer and Surgeon from the Naval Hospital, Subic Bay, arrived on board and were briefed on the remaining casualties to be transferred to their hospital. The deceased were offloaded, followed by the patients. This was completed by 2100 H.

During the following four days the administrative work was nearly impossible. Death certificates were completed on all of the remains transferred to Clark AFB. Communication with the Army Mortuary in DaNang, where the initial remains had been transferred from Repose, was difficult. With the deceased in two so widely separated places the administrative procedures were slowed. On 1 and 2 August additional parts of bodies were recovered and transported to Clark AFB. By noon on Friday, 4 August, I felt that all missing personnel could be assumed dead. The command was advised that the next-of-kin should be so notified.

As I look back now on the disaster, and the calm assured manner in which each member of the Medical Department performed his duty, I am extremely proud. There is little that could have been more efficiently or effectively done. Much of the success in the handling of mass casualties, I attribute to the training and practice that was accomplished under the guidance of the Fleet Training Group in Guantanamo Bay, Cuba in March 1967.

The following are some general comments I have in regard to the disaster:

(1) In a mass casualty accident on the flight deck the Midships Auxiliary BDS (04-123-1-L) is of extremely limited use except as a storage area for first aid supplies. Because of its extremely small size, difficulty in moving a litter patient into the space, and lack of any sheltered casualty holding area, little can be accomplished except immediate first aid, then rapid evacuation via aircraft elevator to the hangar deck and sick bay.

(2) The Forward Auxiliary BDS functioned adequately. It is difficult to move a litter patient into this space. Supplies were more than adequate, except for morphine which had to be obtained from sick bay. The best holding area available, the forecastle, is also extremely difficult to get to with a litter patient.

(3) The Forward BDS functioned well throughout the disaster.

Recommendations:

(1) In a mass casualty accident there is no time to stop and plan. Every ship and station should review its mass casualty handling plan and make sure it works by having adequate drills. Insure that all medical personnel know exactly where they are to be and what they are to do. Assure that triage/treatment areas have the necessary and easily accessible supplies, with special emphasis on: (a) I. V. fluids, especially Ringer's Lactate, along with the administration sets (I strongly recommend the use of angiocaths for all I. V. fluid administration) (b) Adaptic and Kerlex in *large* quantities, for burn dressings (c) Tetanus Toxoid (d) Penicillin-streptomycin combination in tubex form (e) disposable syringes and needles (f) at least six tracheotomy sets (g) one suction machine and six suction set-ups (h) assorted battle dressings (i) Tetracaine, pontocaine, or similar ophthalmic solution; Neosporin, sulfacetamide or similar ophthalmic ointment or solution and eye dressings (j) a cortisone preparation for parental use (k) disposable Foley catheter trays (l) Sparine or similar sedative for parenteral use (m) Demerol in tubex form (n) Phisohex in small squeeze bottles (o) Clagett needles and disposable sterile tubing and bottles for chest suction (p) folding cots or pole litters.

(2) There is a very definite need to make the Forward and After Auxiliary Battle Dressing Stations more accessible to litter patients.

(3) The Midships Auxiliary Battle Dressing Station would be made more useful by installation of wider doors.

(4) Insure that medical supplies are divided equally between the storerooms, and kept at maximum capacity.

(5) It is recommended that the after mess decks be designated as an alternate staging area for mass casualties in the event the sick bay is inoperable. To accomplish this, a moderate sized adjacent space would be needed to store the basic essential medical supplies listed in (1) above. (In addition about 100 pole type litters should be available to hold patients. Stokes stretchers should be returned to the area they came from.)

(6) It is recommended that consideration be given to the placement in Battle Dressing Stations of safes, which could store morphine and Demerol.

(7) All of the dental officers on board Forrestal have had a two day course in handling mass casualties. It is recommended that all dental officers, serving aboard ship, attend such a course.

(8) A set, preplanned treatment for burns greatly facilitates the initial handling of large numbers of these cases. The treatment plan utilized in our disaster was as follows:

(a) Phisohex cleansing of burns. Cold solution was used for pain relief when practical.

(b) Dressing with Adaptic and Kerlex.

(c) I. V. started on all burns above 25% (error on the conservative side).

(d) Foley catheter and hourly urine output monitoring on all burns above 50%.

(e) Tetanus prophylaxis to all burns.

(f) No antibiotic prophylaxis.

(g) Silver nitrate (AgNO3) 0.05% solution burn dressing to all above 30%.

FIRST AID KIT FOR PATROL AIRCRAFT

The following is some recent correspondence relative to First Aid Kits.

From: Commanding Officer, Patrol Squadron FORTY-NINE
To: Commander, U.S. Naval Aviation Safety Center

1. In reviewing the first aid containers aboard squadron aircraft the Flight Surgeon, LT Vukmer, has found them to be inadequate in content. LT Vukmer, along with the safety officer and maintenance officer, has designed and outfitted what is considered to be adequate material and medication for a good first aid kit. The first aid kit, is equipped with material to handle acute problems of airway obstruction, burns, foreign bodies in the eye, sprains, air and sea-sickness, cuts, ear and sinus blocks, and pain from minor problems.

2. The reason for this completeness is that many times crews are in areas where adequate medication is not readily available and self medication is required. Again, on a long patrol an in-flight emergency may necessitate self medication for sinus and/or air sickness. There may be some controversy as to the side effects of some of these drugs, but it is felt that after a short briefing any officer can dispense any or all of the medication from this kit. This squadron has placed similar kits in the duty office and in the maintenance office.

3. All the material and medication is obtainable from any sick bay and replacement is easy. The total cost exclusive of the container itself is $2.97. The container, with other contents, is a standard stock item and can be procured through supply (FSN 6545–656–1093). The first aid kit can be easily attached to any bulkhead in the P3. The contents of the kit can be procured locally and replenished as necessary.

4. It is recommended that the present first aid kit aboard multiengine aircraft be examined along with the contents of this kit with a view toward making a standard first aid kit that will be more beneficial to crewmembers and passengers under most conditions.

From: Chief, Bureau of Medicine and Surgery
To: Commander, Naval Aviation Safety Center, Naval Air Station, Norfolk, Virginia 23511
Ref: (a) COMNAVAVNSAFECEN ltr 42/pn ser 963 of 11 Jul 1967

1. Reference (a) forwarded a letter received from the Commanding Officer, Patrol Squadron Forty-Nine, describing a first aid kit developed by personnel of the Squadron for use in their aircraft. A recommendation was made to review current aircraft first aid kits with a view toward development of a new standard aircraft first aid kit.

2. First Aid Kit, Airplane (FSN 6545–919–6630) is authorized to provide medical material in naval aircraft for self-administered emergency treatment. Such treatment is directed primarily toward treatment of minor injuries. The contents of these kits

INVENTORY OF CONTENTS OF FIRST AID KIT

OUTSIDE

Plastic Pharyngeal Airway

LEFT SIDE

1. Aspirin — Two every three hours for pain
2. Dramamine — One every four hours for air sickness CAUTION: CAUSES DROWSINESS
3. Lomotil — Two every four hours for a total of ten or until diarrhea stops
4. Antiseptic for burns
5. Otrivin nose spray for sinus or ear block
6. Ammonia inhalants
7. Bacitracin ointment for abrasions
8. Tourniquet

RIGHT SIDE

1. Bandaids, eye patch and sterile gauze. Sterile Q-Tip for removal of foreign bodies from the eye
2. Ace Bandage
3. Ear solution for ear pain or drainage
4. Vaseline for burns
5. Bandage for tourniquet or sling
6. Bandage for tourniquet or sling
7. Gauze for use on top of sterile gauze in #1 above

have been chosen to best carry out such treatment, and are the standard items usually found in first aid kits used in varied circumstances and environments. The kit developed by VP–49 contains similar items for use in cases of trauma. The additional items included in the proposed kit are ones used primarily for medical conditions that may arise during flight. An emergency, self-aid sick call kit has thus been developed by the Squadron to care for those conditions which seem to arise most frequently. The items included are those considered by the Squadron Flight Surgeon to be the most appropriate to carry. Another Flight Surgeon may desire different items. Experience has shown that the development of a standard medical treatment kit which would satisfy all is not possible.

3. This Bureau does not object to the development and use of specialized first aid and emergency medical kits by local activities, and interposes no objection to their use either by medical or nonmedical personnel, as long as adequate control is maintained and that adequate training is given to proposed users. The ideas conceived by various medical personnel along these lines are always welcomed, and it is believed should be given dissemination throughout the medical community. It is therefore suggested that the description, including pictures, of the emergency treatment kit developed by VP–49 be published in "Approach" magazine.

4. The incorporation of the pharyngeal airway in the subject kit is especially appropriate for multiplace aircraft such as the P3, and it is believed that its inclusion as a part of standard medical equipment in all multi-place aircraft should be considered by cognizant medical personnel. This item (FSN 6515–660–0046, Airway, Pharyngeal, Plastic, Adult-Child) is readily available from the Medical Material Catalog.

Editorial Comment: As indicated in the above letter, originated in the Bureau of Medicine and Surgery, ideas and recommendations concerning various aspects of operational aviation medicine, aviation physiology training, aeromedical equipment and survival and safety equipment are always welcomed by the Office of Aerospace Medicine. Guarantees cannot be given that all ideas and recommendations will be adopted as official policy or will result in changes in standard equipment, but it can be assumed that they will be conscientiously reviewed and considered, and appropriate action taken.

Another point in the Bureau's letter which deserves special note. Whereas it is indicated there is no objection to the development of specialized first aid and medical kits for use by either medical or nonmedical personnel, the conditional statement accompanying that endorsement is the really pertinent one. A real need must exist to warrant the placing of such a kit, and there must be some responsible person or persons to oversee its utilization. Self-medication can be very dangerous, not only to pilots but also to ground and maintenance personnel who are operating machinery and working around machines. Also self-medication tends to encourage an individual to delay going to sick call; an action which could have serious consequence. Such action involving self-aid for an injury, particularly in a civilian worker, can cause considerable difficulty in determining disability and compensation in the event complications or future sequelae result.

The main point to be stressed is the responsibility the Flight Surgeon assumes when he makes available materials for medical self-aid and treatment. He must assure himself that personnel having access to the materials have been thoroughly indoctrinated in their use, and that they use them as an adjunct prior to seeking professional medical attention.—AeroMed, BuMed.

NEW DEVELOPMENTS IN AIRCREW PROTECTIVE EQUIPMENT

The Aerospace Crew Equipment Department, now a component of the Naval Air Development Center, Johnsville, but still located at its previous site at the Philadelphia Naval Base, has been heavily involved in direct support test, and evaluation of aircrew personal survival equipment. The following are recent highlights in this area.

The Aerospace Crew Equipment Department (ACED) has developed a new flight glove constructed with a non-slip, washable leather palm and a fire resistant, non-melting fabric back called 'Nomex.' The palm portion of the glove provides dexterity and 'feel' required by the aviator, while the fabric provides superior comfort and fire protection compared to other gloves. Initial supplies of the new glove are in use by pilots in Vietnam.

In response to an urgent need for increased life preserver buoyancy and flotation positioning improvement due to the increased weight and bulk of personal survival equipment now being carried by pilots in combat areas, ACED has developed a new

life preserver designated the LPA–1. This preserver has much less bulk and greater wearing comfort compared to other types while uninflated. When in use, it provides 65 lbs. of buoyancy plus automatic head positioning for an unconscious survivor in the water. The LPA–1 is compatible with the SV–2 survival vest, also developed in response to Vietnam requirements, which permits optimum location and stowage of the full inventory of personal survival equipment for aviators in combat. The new preserver is now under procurement for the Fleet and will eventually be an across-the-board replacement for all life preservers, particularly the Mae-West which has only 20 lbs. of buoyancy.

ACED is heavily involved in development, test, and evaluation of modifications to current ejection seat escape systems incorporating rocket boosters to achieve zero airspeed, zero altitude escape capability for our fighter and attack aircraft. One novel system, which is being incorporated in the propeller-driven A–1 Skyraider aircraft and is under development for other aircraft types, is the YANKEE Escape System which actually pulls the pilot from his disabled aircraft by use of a rocket, rather than by ejection of the seat in the customary fashion.

A new light weight rescue net that reduces helicopter rescue hover time in both land and sea rescue situations has been developed and is in final stages of evaluation for SAR mission requirements. Its simplicity of operation, light weight, small bulk, ease of stowage, and lack of complicated maintenance requirements make it ideal for use in all types of helicopters. The same manufacturer has also developed a jungle-penetration rescue seat which provides head and shoulder protection for the rescuee who must be hoisted up through heavy foliage. This device is also undergoing final evaluation by ACED in the jungle cover environment of the Philippines to test its effectiveness for use in the forest cover in Vietnam.

Along with its many activities in the research, development, test, and evaluation of aircrew equipment, one of ACED's more basic research accomplishments by the Life Sciences Research Group, which has generated considerable interest, is work currently underway in pulmonary physiology. A new method of presenting respiratory flow-volume date in pulmonary function assessment has been devised which involves the placement of an accurate loop tracing on an absolute flow-volume reference grid in one operation. This produces a graphic representation of all lung volumes and peak flows at all volumes. This method, currently being evaluated in cooperation with the cardiopulmonary laboratory at the Naval Hospital, Philadelphia, is expected to aid in the diagnosis of pulmonary disease by means of characteristic flow-volume loop signatures, as well as greatly facilitate communication of the results of pulmonary function tests.

CONFERENCE ON SPECIALTY TRAINING IN AEROSPACE MEDICINE

The Assistant Chief of the Bureau of Medicine and Surgery for Aerospace Medicine, CAPT W. M. Snowden, MC USN, represented the Surgeon General at the Conference which was hosted by the Ohio State University and chaired by the Secretary of the American College of Preventive Medicine, Harold V. Ellingson, M. D. The Naval Aerospace Medical Institute was represented by CDR M. C. Carver, MC USN, Assistant Head, Training Department.

This was a working conference on the requirements for Residency Training in Aerospace Medicine and was attended by representatives of the U.S. Army; U.S. Navy; U.S. Air Force; National Aeronautics and Space Administration; Federal Aviation Administration; Commercial Airlines and the Aerospace Industries. CAPT Snowden presented an address entitled, "Training and Utilization of Naval Flight Surgeons" together with the film "Doctor on the Flight Deck". CAPT Snowden noted that of the Naval Medical Officers certified in Aerospace Medicine there are at present thirty-two still on active duty. There is each year a total of eighteen Navy Flight Surgeons in the residency training program. Six are in schools of Public Health for one year and the remainder assigned to the Naval Aerospace Medicine Institute for two years.

The current output of six qualified specialists per year is considered adequate for the Navy's needs at this time. CAPT Snowden made recommendations to the American College of Preventive Medicine aimed at facilitating the taking of Board Examinations by Navy residents, who have often in the past found themselves deployed overseas and unable to return at the time the examinations were administered.

SAFE SYMPOSIUM

The Fifth Annual Survival and Flight Equipment Association (SAFE) Symposium was held in San Diego, California 26–28 September 1967. Navy representatives from various commands concerned with survival equipment were in attendance. Dem-

onstrations were provided by several industrial representatives of the latest developments in aerial and hand-held smoke and candle signal flares. Included in the demonstrations were several pen gun-type flare kits and a radar rocket flare system which disperses chaff which is visable on a radar screen at distances up to 160 miles.

Other presentations covered oxygen systems, personal equipment, aircraft escape and evacuation, search and rescue, and aviation physiology and accident prevention. Attendees visited the Aerospace Museum at Balboa Park, the Physiology Training Unit at Miramar, and other activities in the San Diego area. Mr. R. J. O'Leary of United Airlines spoke at the banquet meeting on "The Mark II Human, Who Needs Him?

SAFE's address is Aerospace Museum, Balboa Park, San Diego, California 92134. Executive Advisor is Mr. Jim Johnson.—Submitted by LCDR K. H. Dickerson, MSC USN, Naval Air Systems Command Headquarters (AIR–5314B).

XVI INTERNATIONAL CONGRESS ON AVIATION AND SPACE MEDICINE

The Surgeon General and the Bureau of Medicine and Surgery were represented at the XVI International Congress on Aviation and Space Medicine held in Lisbon, Portugal, 11–15 September 1967 by CAPT W. M. Snowden, MC USN. This was the largest International Congress ever convened and the Navy participated by presenting a paper entitled "Training and Utilization of United States Naval Flight Surgeons". Additionally, with the support of the State Department, four films were shown daily during this Congress. The films presented were "Doctor on the Flight Deck", "Fit to Fly", "Aircraft Accident Investigation", and "SEALAB II".

EDITOR'S SECTION

THE WATER WE DRINK

Arthur G. Schoch MD, The Schoch Letter, Medical Arts Building, Dallas, Texas.

We all drink water from the cradle to the grave, but we pay no attention to what kind of water we drink. The food we eat changes with age, but not the water. In infancy, food is milk and cereal. In childhood, the meat and vegetable era starts, together with peanut butter and jelly sandwiches, but the water is the same. During adolescence and young adult life, the quality of the food consumed is very variable, the amount available of utmost importance, but the water is the same. Starting at about middle age, the kind of food we eat assumes great importance and people learn about calories, chloesterol, proteins and carbohydrates, shuffle these about in a most indiscriminate manner, depending on the dietary fad in vogue, yet the water we drink remains the same.

From middle life on, we (doctors as well as patients) develop an even greater interest in diet as it pertains to the degenerative diseases, particularly those affecting the cardiovascular system (e.g. heart trouble (hardening of the arteries, high blood pressure) and bone structure (e.g. osteoporosis or softening of bone, and arthritis). This is the era of dietary supplements, vitamins, minerals, and hormones, commonly used in maintenance therapy, and yet we continue to ignore the water we drink.

To drink hard water is good. To drink soft water is bad. At least this is the inescapable inference that we draw from independent studies done in Sweden, Great Britain, and the United States, all indicating that cardiovascular disease and death, calcification of the aorta and great vessels, and osteoporsis are significantly more prevalent where the drinking water is soft than in areas where the drinking water is hard. This is the only known environmental influence on the many-faceted "aging problem."

No one seems to have clearly defined hard water and soft water. Very roughly speaking, the hardness of 100 p.p.m. (parts per million parts of water) is about average. Roughly, half of the United States water supplies, running from 100 p.p.m. to over 200 p.p.m. have hard water. The other half, having less

From The Penicillin Research Center—Southwestern Medical Foundation—Dallas.

than 100 p.p.m. of hardness and the majority below 50 p.p.m., are regarded to have soft water.

Hardness in drinking water must be divided into (1) permanent hardness, from the calcium and magnesium cations in solution, and (2) temporary hardness as measured by the amount of bicarbonate ions in solution. Boiling the water, for example to make tea or coffee, removes the desirable temporary hardness (HCO_3) by forming in the insoluble calcium carbonate which precipitates. This leaves a certain amount of hardness in the teakettle in the form of precipitate, but the permanent hardness remains (soluble calcium and magnesium salts) for us to consume, along with our tea or coffee. Drink water from the spigot and you get the total hardness therein.

The water we drink, in the main, is "purified" by (1) softening it, (2) chlorinating it, (3) filtering it, and (4) fluoridating it. In this process, the sodium content of our drinking water is never reduced, and is often increased, for instance by the lime and soda treatment for softening water. The sodium content of drinking water is the only facet of water's mineral content to which medicine, in general, has paid much attention. This is due to the simple fact that the higher the sodium intake from any source, the greater the retention of body fluid. In most instances a low sodium diet in a patient with edema accomplishes practically nothing if the patient continues to drink city water. So what is the solution? The patient subsists on bottled distilled water, sodium free, of course, but also free of the calcium, magnesium, bicarbonate, and fluoride ions so very essential generally, and particularly in this type of patient.

Fluoridation of city water supplies consists of adding a homeopathic amount of sodium fluoride so that the water contains from 1 to not over 2 p.p.m. of fluoride. Its sole purpose is to prevent tooth decay in children. For the adult, this meager amount of fluoride in the drinking water is totally inadequate to significantly prevent osteoporosis developing in later adult life, particularly in women. A reasonable guess would be that ten times this amount of fluoride would be required for adults. A most impressive case report recently indicated a cure of advanced osteoporosis, a complication of cortisone therapy, by the oral administration of 100 mg. of sodium fluoride daily for a bit over a year, without stopping the causative prednisolone (cortisone) maintenance therapy.

Viewed from the standpoint of the anti-fluoridationists, may their bones soften and they shrink in stature, this colossal amount of sodium fluoride taken internally would certainly result in cancer or sudden death from fluoride poisoning—but it didn't. The patient got out of bed and went back to work.

The Bottled Water We Drink

Many of us drink little or no water, per se. How many times have you heard a person say, "I never drink water?" These people get their necessary daily amount of H_2O in the form of milk, coffee, tea, soda pop, beer, wine, or highballs. Milk, of course, is high in calcium and magnesium and noxious sodium (disallowed on a salt-free diet). The water used in bottled drinks, such as beer, soda pop, et cetera, is all processed, and very carefully so, in bottling plants, in order that a bottle of beer or Coca Cola TASTES THE SAME in New York City as in Kalamazoo, the same in Alaska as in Honolulu. The American public demands this consistency in taste, and they get it.

Bottled mineral water, to mention one specifically, Mountain Valley Water from Hot Springs, Arkansas, is available from coast to coast. Although most of its hardness is temporary and can be boiled out, it contains no fluoride, and is practically sodium free. It is an excellent hard drinking water.

Do It Yourself

If one would like to add calcium, magnesium, and fluoride salts to distilled water, in order to have hard water containing fluoride, one merely has to add, in proper amounts: gypsum (calcium sulfate); bischofite (magnesium chloride); and sodium fluoride. These simple inexpensive chemicals are readily obtainable from your pharmacy. One milligram per liter equals one part per million. Since the atomic weight of calcium is 40, and the molecular weight of calcium sulfate is 172, in order to get 100 p.p.m. of calcium one adds 434 mg. per liter of gypsum. To add 24.3 p.p.m. of magnesium, one adds 203 mg. of bischofite per liter of water. If you wish this water to have 10 p.p.m. of fluoride, add 22 mg. of sodium fluoride per liter of water. This in round numbers would yield drinking water that contains 100 p.pm. of calcium ions, 25 p.p.m. of magnesium ions, and 10 p.p.m. of fluoride ions. This would be a fine drinking water for the old folks, but far too much fluoride for children, because it would almost certainly result in mottled enamel. It has five to ten times as much fluoride as approved water supplies, which contain from 1 to 2 p.p.m. in order to prevent tooth decay without producing the mottled enamel. This would yield an ideal hard water for drinking purposes (with or without the fluoride). The total

permanent hardness would be approximately 125 p.p.m., two or three times as hard as most of the water consumed in these United States. The calcium and magnesium ion content would be almost identical to that which we find in human body fluids (serum calcium 100 p.p.m., and serum magnesium 22-25 p.p.m.), and, therefore, isotonic with the blood serum.

Why We Soften The Drinking Water

Nearly all cities, large and small, have a water-treating plant which provides "improved water" to all consumers for various purposes. This water is softened, cleared of silt by filtration, cleared of bacteria by chlorination, and minutely fluoridated, if the anti-fluoridationists can be outvoted in the community. Why soften the water when hard water for human consumption is far more desirable than soft water? It all started about fifty years ago with the fatuous and myopic thought that soft water requires less soap (dollars saved for commercial laundries) and that boiler pipes did not form scale (saving money for industry), without thought to the good or bad of it when people drink it. Therefore, today the man in charge of the water you drink is almost invariably an engineer, completely unqualified, by virtue of training, to give much thought to the health of the people who drink the water that he "manufactures."

The water that your city supplies you, you drink. You also use it to wash the dishes and clothes, water the grass and garden, bathe with it and flush the toilets. This water goes into your stomach, down the sewer or on the yard.

The industrial use of water, particularly if one includes large-scale irrigation projects, is tremendous, whereas the water we drink is infinitesimal in amount, by comparison. That is why we are forced to drink water treated primarily for industrial use with an inadequate mineral content.

To make this present paradox even more ludicrous, consider the general demise of boiler plants in these United States. We no longer use steam engines as locomotives on railroads. Electrical power in the main is generated at large dams rather than coming from the local power plant which formerly made use of steam turbines (and boilers) to turn the electric generators. The great plague of boiler scale in major industry has largely vanished like the horse and buggy.

A Dual Supply of Electric Power—Why Not Water?

If you are an urban dweller in the United States, and most of us are, public utilities are essential to your daily existence. The power and light company furnishes at least two types of electrical power to your residence: one a single phase 110 volt source of electric current on which most household utilities, and particularly electric lights, operate upon. In addition, they will furnish triple phase 220 volt power for heavier electrical appliances, particularly air conditioning units. If your power and light company can furnish dual service, why not your water company? Why could we not all have water of maximum benefit to human consumption, and another source of water, crude water, for the lack of a better term, to water the grass and flush the toilet, et cetera? Water from the old rain barrel, years ago, was used by the fastidious females to wash their hair, wiggle tails and all, but they didn't drink it. In those days the drinking water was hard; it came from a well, or from a spring filtered through limestone with a high content of calcium and magnesium ions!

Drink hard water all of your life, because it is good for your teeth, your bones, your heart, and for your blood vessels. And to steal an addendum from television commercials—"It tastes good too."

(The bibliographies may be seen in the original article.)

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